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NEWS 3. Jan 29. ESTA has been reloaded and moves to member in NEWS 4. Feb 01. DKH IT now produced by FIZ Karlsruhe and has a new update frequency

/ / / / ۰, 1-69 10 Access via Tynmet and SprintNet Fliminated Effective 3/31/02

1111 **3** Mar 08 Gene Names now available in BIOSIS

1115 Mar 22 TONLIT no longer available

/ / / / x Mar 22 TRCTHERMO no longer available

V.F.W.S 9 Mar 28 US Provisional Priorities searched with P in CA CAplus and USPATEULL

NEWS 10 Mar 28 LIPINSKI CALC added for property searching in REGISTRY .\pr 02 PAPERCHEM no longer available on STN | Use PAPERCHEM2 instead

ンナジェー .\pr 08 "Ask CAS" for self-help around the clock

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VEWS 15 \pr 19 .\pr 09 US Patent Applications available in IFICDB, IFIPAT, and IFIUDB

ンドシェニュ .\pr 22 Records from IP.com available in CAPIA'S, HCAPIA'S, and ZCAPIA'S

.\pr 22 BIOSIS Gene Names now available in TONCENTER

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http://www.a.cas.org/ONLINE/STN/STNOTES stnotes27 pdf

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2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA 47 REFERENCES IN FILE CAPILIS (1967 TO DATE) APLUS, TONCENTER, USPATFULI ENTRY SESSION SINCE FILE 7.10 7.31 COTAL

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- STRUCTURE DIAGRAM IS NOT AVAILABLE
- 2729 REFERENCES IN FILE CA (1967 TO DATE)
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- ST30; CN GAL-BETAL3-GALNAC-ALPHA2,3-SLMD/LTRANSFERASE (RATIOTON) FALD-GALNAC-, ALPHA 2,3-SLALY LTRANSFERASE (PIG CI ON)
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SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 Apr 13) 276-15 11590-8 II Estragen receptor-mediated activation of the serum response element in MCF-7 cells through MAPK-dependent phosphorylation of Elk-1. L18 ANSWER LOF II Journal code: HIV; 2985121R, ISSN: 0021-9258 MAPK-dependent phosphorylation of Elk-L MEDLINE DUPLICATE 1

LIS ANSWER ZOUTH MEDITAL

- 11 High glucose-enhanced mesangial cell extracellular signal-regulated protein kinase activation and alphal(IV) collagen expression in response to endothelin-1 role of specific protein kinase C isozymes.
- SO DLABITES (2001 Oct) \$0 (10) 2376-83. Journal code 1/8N, 0372763 ISSN: 0012-1797

NSWIR 3 OF II MEDIAN

DUPLICATE 3

- 11 I popolysaecharide activation of the MEK-ERK1 2 pathway in human menocytic inducing Elk-1 phosphorylation and Egr-1 expression cells mediates tissue factor and tumor necrosis factor alpha expression by
- SO BLOOD, (2001 Sep 1) 98 (5) 1429-39 Journal code 1/80, 7603509 ISSN: 0006-4971

1.18 ANSWER 4 OF 11 MEDIANE

PULLY OF ILV

- epidermal growth-factor receptor The anthocyanidins cyanidin and delphinidin are potent inhibitors of the
- SO JOURNAL OF AGRICULTURAL AND FOOD CHEMISTRY. (2001 Feb) 49 (2) 958-62. Journal code: H3N: 0374755-1SSN-0021-8561.
- ANSWER 5 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
- T1 1 PS activation of the MEK-FRK1 2 M APK pathway in human monocytic cells induces I gr-1 gene expression. Role in the induction of inflammatory mediators
- SO Blood. (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 607a. print Hematology San Francisco, California, USA December 01-05, 2000 American Society of Meeting Info 42nd Annual Meeting of the American Society of Hematology 150t-9000 NSSI
- 118 ANSWER 6 OF 11 CAPIL'S COPYRIGHT 2002 ACS
- 11. C.F.BPBeta and Elk-1 synergistically transactivate the c-fos serum response
- SO BMC Cell Biol (2000), 1. No pp. given CODEN BCBMAY, ISSN: 1471-2121 URL http://www.biomedcentral.com/content.pdf/1471-2121-1-2.pdf/

LIS ANSWER 7 OF II

SO CHILLIAR SIGN WHING (1999 Aug) 11 (8) 575-80 protein expression Journal code AVB, 8904683 ISSN: 0898-6568

11 Protein phosphatase 2.\(\text{Suppresses MAP kinase signalling and ectopic}\)

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LIS ANSWER & OF TE MEDITAL

DUPLICATE 6

- 11 p38 Mitogen-activated protein kinase mediates the transcriptional induction of the atrial natriuretic factor gene through a serum response
- element. A potential role for the transcription factor ATF6
 SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Aug 7) 273 (32) 20636-43 Journal code HIV, 2985; 218 1888; 6021-9258

118 ANSWER 9 OF 11 MEDI INF

DUPLICATE 7

11 Growth factor-induced transcription via the serum response element is inhibited by cyclic adenosine 3',5'-monophosphate in MCF-7 breast cancer

SO TYDECEINOLOGY, (1997 Jun) 138 (6) 2219-26 Journal code: EGZ: 0375040. ISSN: 0013-7227.

L18 ANSWE 2 10 OF 11 MEDLINE

8 JUV OURLIN

TI Functional role of extracellular signal-regulated protein kiruses in gastric acid secretion.

Journal code: 3U8; 0370511, ISSN: 0002-9513. MIFRICAN JOURN M. OF PHYSIOLOGY, (1997 Dec., 273 (6 Pt.1) G1263-72

L18 ANSWER 11 OF 11 MEDIANE

DUPLICATE 9

TI Urea-inducible Egr-1 transcription in renal inner medullary collecting duct (mIMCD3) cells is mediated by extracellular signal-regulated kinase activation.

STATES OF SO PROCEDINGS OF THE NATIONAL ACADEANY OF SCIENCES OF THE CATLED

Jeurnal code: PV3; 7505876, ISSN: 0027-8424 AMIER CA. (1996 Oct 1) 93 (20) 11242-7.

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1 18 ANSWER 9 OF 11 AFDI ICE

DUPLICATE 7

ACCESSION NUMBER: 97307662 MEDITAL

DOCUMENT NUMBER 97307662 PubMed ID 9165004

element is inhibited by cyclic adenosine Growth factor-induced transcription via the serum response

1',5'-monophosphate in MCF-7 breast cancer cells.

COMMENT MOH... IV Lowe W.L. Jr; Fu R; Banko M Comment in: Endocrinology 1997 Jun; 138(6):2217-8

CORPORATE SOURCE: lealthcare System and Northwestern University Medical Department of Medicine, Veterans Administration Chicago

SOURCE School, Chicago, Illinois 60611, USA wlowe anwuedu ENDOCRINOI OGY, (1997 Jun) 138 (6) 2219-26

Journal code: EGZ, 0375040. ISSN: 0013-7227

PUB. COUNTRY United States

Journal; Article: (JOURNAL, ARTICLE)

LANGUAGE English

FILE SHOW EVE Abridged Index Medicus Journals; Priority Journals

INTRY MONTH 99706

ENTRY DATE: Last Updated on STN: 20000303 Entered STN: 19970630

Fintered Medline: 19970617

AB. The effect of increased intracellular cAMP on MCF-7 breast cancer cell effect of cAMP on growth factor-induced ERK activity in MCF-7 cells was either 1 or 10 microM forskolin decreased cell growth by 17% and 68%. examined. Treatment with either insulin-like growth factor I (IGF-I) or mediated by inhibition of the activity of extracellular signal-regulated growth by 29%. To determine whether this effect of cAMF on cell growth was respectively, whereas treatment with 250 microM 8-CPT-CAMP decreased cell analog. Compared to cells maintained in control medium, treatment with of adenylate cyclase, growth v as examined by treating cells with either ferskolm, an activator and 2 (ERK1 and -2), two mitogen-activated protein kinases, the or 8-[4-chlorophenylthio]-cAMP (8-CPT-cAMP), a cAMP

variety of growth-premoting events, was examined. For these assays, MCF-7 treatment with forskolin partially inhibited the activation of EIK mereased using 8-CFT-cAMP, similar results were obtained. SRE activity is observed using 1 microM forskolin. When intracellular cAMP levels were luciferase activity by approximately 75%. An intermediate effect was element (SRF), a DNA promoter element whose activity is regulated by a effect on IGF-1- or EGF-induced FRK activity in cells treated with growth inhibited by increased intracellular c.VMP generated by pretreatment of the merease in ERK1 and -2 activity. This effect of IGE-I and EGF was not sites cloned 5' to a thymidine kinase promoter and the luciferase gene UNS-TK-Luc, a plasmid that contains two Gal4 DNA recognition of cells with 10 microM forskolm decreased IGF-I- and EGF-stimulated gene that contains the SRE cloned 5 to a minimal thymidine kinase factor-mediated effects, its effect on the activity of the serum response factor for 30 mm. To determine whether eAMP inhibits other growth cells with 10 microM forskolm. Similarly, 10 microM forskolin had no epidermal growth factor (EGE) for 10 min stimulated a 4- to 8-fold -1 by IGF-I and EGF These data demonstrate that in MCF-7 breast expresses a Gal4 Elk-1 fusion protein and 1 When MCF-7 cells were cotransfected with a vector that factor, included among the ternary complex factors is Elkdependent upon the activation by phosphorylation of a ternary complex mereased pTK81-SRE-I ue activity in a dose-dependent fashion. Pretreatment promoter and the luciferase gene. Treatment with either IGF-I or EGF cells were transfently transfected with pTK81-SRE-Luc, a luciferase fusion

through inhibition of specific growth factor-induced effects, including gene transcription

1.18 ANSWER 7 OF 11 MEDIANE DUPLICATE 5

AND ANSWER 1 MEDIAN MEDIANE.

I RK-independent pathway. Finally, these data indicate that the effect of

mereased intracellular eAMP on breast cancer growth may be mediated

but it inhibits growth factor-induced transcription. Taken together with

the effects of cAMP on IGF-1- and EGF-induced Elk-1

cancer cells, e.AMP has no effect on IGF-1- or EGF-induced ERK activity,

activation, these data suggest that the effect of eAMP on SRL activity

occurs distal to I RK activation, possibly via inhibition of an

ACCESSION NUMBER: 1999360900 MEDIANE
DOCUMENT NUMBER: 99360900 PubMed ID: 10433518
ETHE Protein phosphatase 2.A suppresses MAP kinase signalling and ectopic protein expression.
AUTHOR Chang H. Brautigan D.L.

Charlottesville 22908, USA.
SOURCE CELLULAR SIGNALLING, (1999, Aug.) 11 (8) 575-80
Journal code. AVB, 8904683-ISSN; 0898-6568

PCB COUNTRY

FNGLAND United Kingdom

CORPORATE SOURCE: Center for Cell Signalling, University of Virginia

Journal, Article, (JOURNAL ARTICLE)
FANCIUAGE English
FILE SEGMENT Priority Journals
FNTRY MONTH 199910
FATRY DATE: Faterial STN 19901026

FNTRY DATE: Entered STN 19991026 Last Updated on STN 19991026 Entered Medline: 19991012

VB. Signalling by MAP kinase was examined in COS-7 cells by transiently.

expressing a transcription reporter system plus epitope-tagged protein phospha ase 2A catalytic subunit [(HA)3-PP2Ac]. Transactivation of a luciferase gene by GAL4-Blk-1 in

serum-stim flated cells was reduced 20-fold by co-expression of wild type (HA)3-I P2Ac. This reduction of MAP kinase signalling required specific type-2A phosphatase activity, because the effects were not minicked by co-expression of either a mutated, inactive (HA)3-PP2Ac or wild-type PP1Cde ta. Expression of (HA)3-PP2Ac was severely restricted by its own activity because 3-fold more inactive (HA)3-PP2Ac was produced. In a differen assay the kinase activity of FLAG-ERK2 was 4-fold lower when co-transfected with (HA)3-PP2Ac, compared to controls. Unexpectedly, mRNA of the reporter constructs were nearly eliminated by even low level expression of (HA)3-PP2Ac in either COS7 or HFK293 cells. The results show that PP2A activity is strictly regulated and can be a limiting factor in ectopic expression of various proteins.

L18 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001/792927 CAPLUS

TITLE: C EBPBeta and Elk-1 synergistically transactivate the

e-fos serum response element

AUTHOR(S) Hanlon, Mary: Bundy, Linda M.; Sealy, Linda

CORPORATE SOURCE: Department of Molecular Physiology and Biophysics, Vanderbilt University School of Medicine, Nashville,

VS.1.X.L

SOURCE BMC Cell Biol. (2000), 1, No pp. given

CODEN: BCBMAY; ISSN: 1471-2121

URL: http://www.biomedcentral.com/content/pdf/1471-2121-1-2.pdf

PUBLISHER: BioMed Central Ltd.

DOCUMENT TYPE: Journal: (online computer file)

LANGUAGI. English

AB Background: The serum response element (SRE) in the c-tos promoter is a converger ce point for several signaling pathways that regulate induction of the c-fe's gene. Many transcription factors regulate the SRE, including serum response factor (SRE), ternary complex factor (TCE), and CCAAT enhancer binding protein-beta (CEBP, beta.). Independently, the TCEs and CEBP, beta, have been shown to interact with SRE and to respond to Ras-dependent signaling pathways that result in transactivation of the SRE. Due to these common observations, we addressed the possibility that CEBP, beta, and Elk-1 could both be necessary for Ras-stimulated transactivation of the SRE. Results: In this report, we demonstrate that Elk-1 and CEBP, beta, functionally synergize in transactivation of both a Cal4 reporter plasmid in concert with

Gal4-SRF and in transactivation of the SRE. Interestingly, this synergy is only obsd. upon activation of Ras-dependent signaling pathways. Furthermore, we show that Elk-1 and C EBP, beta, could interact both in an in vitro GST-pulldown assay and in an in vivo co-immunorptin, assay. The in vivo interaction between the two proteins is dependent on the presence of activated Ras. We have also shown that the C-terminal domain of C EBP, beta, and the N-terminal domain of Flk-1 are necessary for the proteins to interact. Conclusions: These data show that C I BP, beta, and Flk-1 syr ergize in SRF dependent transcription of both a Gal-4 reporter and the SRE. This suggests that SRF, TCF, and C EBP, beta, are all necessary for maximal induction of the c-fos SRE in response to mitogenic

REFERENCE COUNT signaling by Ras 36 THERE ARE 36 CITED REFERENCES AS ALL ABLE FOR

RECORD AND CITATIONS AVAILABLE IN THE RE FORM VI

OCCUMENT NUMBER PREV200100322077 T18 ANSWER 5 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTEACTS INC

induction of inflammatory mediators. monocytic cells induces Egr-1 gene expression. Role in the LPS activation of the MEK-ERK1 2 MAPK pathway in human

VI THOR(S) P (1), Mackman, N (1) Guha, M. (1), O'Connell, M. (1); Hollis, A. (1); McGovern

CORPORATE SOURCE: (1) The Scripps Research Institute, San Diego, CAUSA

Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp.

Meeting Info., 42nd Annual Meeting of the American Society

01-05, 2000 American Society of Hematology 188N 0006-4971. of Hematology San Francisco, California, USA December

Conterence

DOCT MENT TYPE

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SUMMIARY LANGUAGE English

VB 1 PS induces human monocytes to express many proinflammatory mediators. mediate LPS induction of TNF and TF gene expression. In this study, we (TF) We have shown that Egr-1 and NF-kappaB Rel transcription factors One of these sites (SRE4) bound a LPS-inducible complex that contained SRF including the cytokine TNF and the procoagulant molecule tissue factor Eppal3-dependent transcription. In contrast, PD98059 strongly inhibited investigated the role of the MEK-ERK1-2 MAPK pathway in LPS induction of and Elk-1 | LPS induced phosphorylation of Elk mediated by a approximately approximately that contained three SRE sites (SRE3-5). I PS induction of Egr-1 expression, LPS induction of the Egr-1 promoter was manner PD98059 did not affect LPS-induced nuclear translocation of PD98059 reduced LPS induction of TNF and TF expression in a dose-dependen TNF and TF gene expression in human monocytic cells. The MEK1 inhibitor NF-kappaB Rel proteins and minimally affected LPS induction of

-1 and increased the functional activity of a GAL4

-FIK-TTA changene protein. Activation of Elk-1 was

inhibited by PD98059. Our data indicate that LPS activation of the

of the TNF and TF genes in human monocytic cells. activation of pre-existing NF-kappal3 Rel complexes, for maximal induction MFK-FRK1 2 pathway and Egr-1 gene expression is required, together with

1.18 ANSWER 4 OF 1 ACCESSION NUMBER 2001285773 MEDITAL MEDI INE

DOCTAIFATATAIBER 21162838 PubMed ID: 11262056

The anthocyamdins cyanidin and delphinidin are potent

inhibitors of the epidermal growth-factor receptor Meiers S. Kemeny M. Weyand U. Gastpar R. von Angerer F.

CORPOR VIF SOURCE: Department of Chemistry, Division of Food Chemistry and Frwin-Schroedinger-Strasse 52, 67663 Kaiserslautern, Environmental Toxicology, University of Kaiserslautern.

Germany

301.103 (2) 958-62. JOURNAL OF AGRICUTIUR AL AND FOOD CHEMISTRY, (2001 Feb. 49)

Journal code: H3N; 0374755, ISSN: 0021-8561.

PUB COUNTRY: United States

Journal: Article; (JOURNAL ARTICLE)

LINGUAGE English

FILE SEGMENT Priority Journals

ENTRY MONTH 200105

ENTRY DATE Fntered STN: 20010529

List Updated on STN 20010529

Entered Medline: 20010524

VB. The aglycons of the most abundant anthocyanins in food, cyanidin (cy) ind overexpressing the epidermal growth-factor receptor (EGFR). The glycosides delphinodir (del), were found to inhibit the growth of human tumor cells anthocraridins ey and del are potent inhibitors of the EGFR, shutting off in the concentration range where growth inhibition was observed. Thus, the inhibited the growth of the human vulva carcinoma cell line A431. constituer ts. substantially to the growth-inhibitory properties of these natural food downstream signaling cascades. These effects might contribute investigated by measuring the phosphorylation of the transcription factor anthocyamdin in grapes, was less active. The aglycons preferentially in vitro in the micromolar range, whereas malvidin (mv), a 'ypical activation of the GAL4-Elk-1 fusion protein -1 fusion protein. We kinase bataway dependent phosphorylation of a GAI 4-FIK lucifer; se reporter gene construct whose expression is controlled by MAP influence of anthocyanin treatment on downstream signaling cascades was ey-3-gal and my-3-gle were inactive up to 100 microM. In intact cells the cells, was potently inhibited by cy and del. My and the glycosides micro\ The tyrosine kinase activity of the FGFR isolated from A431 glucoside (mv-3-gle, oenin) did not affect tumor cell growth up to 100 eyanidin-3-beta-D-galactoside (ey-3-gal, idaem) and malvidin-3-beta-D-Elk-1. A431 cells were transiently transfected with a found that cy and del inhibited the

1.18 .NSW :R 3 OF 11 MEDIANE DUPLICATE 3

ACCESSION NUMBER 2001491800 NEDITAL.

DOCI VII VII VI VI VIBER 21411459 PubMed ID, 11520792

numan monocytic cells mediates tissue factor and tumor Lipopolysaecharide activation of the MEK-ERK1-2 pathway in

phosphorylation and Egr-1 expression. necrosis factor alpha expression by inducing Elk-1

M.THOR: (iuha M; O'Connell M A; Pawlinski R; Hollis A; McGovern P;

CORPORA' E SOURCE: Yan S F; Stern D; Mackman N Department of Immunology. The Scripps Research Institute.

CONTRACT NUMBER La Jolla, CA 92037, USA III.48872 (NIII.I3I)

BLOOD, (2001 Sep 1) 98 (5) 1429-39

Journal code: A80; 7603509 ISSN: 0006-4971.

PUB CCUNTRY United States

Journal: Article; (JOURNAL ARTICLE)

LANGU YGE English

ETE SEGMENT Abridged Index Medicus Journals: Priority Journals

ENTRY MONTH Fintered Medline 20011004 Last Updated on STN 20011008 Entered STN 20010906

VB. I ipopolysaecharide (LPS) induces human monocytes to express many reduced IPS induction of IF and TNF-alpha expression in a dose-dependent including nuclear factor (NF)-kappaB Rel proteins and I gr-1. In this promflammatory mediators, including the procoagulant molecule tissue response factor and Elk-1, LPS stimulation transiently pathway in I PS induction of TF and TNF-alpha gene expression in human expression is required for maximal induction of the TNF-alpha and TF genes induced phosphorylation of Elk-1 and increased the expression. In addition, mutation of the Egr-1 sites in the TF and experiments LPS induction of Fig.-1 expression preceded induction of TF pathway strongly inhibited LPS induction of Egr-1 expression. In kinetic EappaB-dependent transcription. In contrast, PD98059 and dominant-negative study, the role of the MFK-ERK1-2 mitogen-activated protein kinase (MAPK) in human monocytic cells. MFK-FRK1/2 pathway. The data indicate that LPS induction of Fgr-1 gene functional activity of a GAL4-Elk-FTA chimeric protein via the mutants of the Ras-Raff-MEK-ERK (extacellular signal-regulated kinase) manner PD98059 did not affect LPS-induced nuclear translocation of 1F and TNF-alpha genes are regulated by various transcription factors. factor (TF) and the cytokine tumor necrosis factor alpha (TNF-alpha). The 3 SRF sites, which bound an LPS-inducible complex containing serum was demonstrated that LPS induction of the Egr-1 promoter was mediated by NI-kappaB Rel proteins and minimally affected LPS induction of monoextic cells was investigated. The MAPK kinase (MFK)Uinhibitor PD98089 INF-alpha promoters reduced expression of these proinflammatory genes. I.

DOCUMENT NUMBER I 18 ANSWER LOF II ACCESSION NUMBER 2001287501 VIII DI INI 21192198 | PubMed ID: 11145955 MEDIANE DUPLIC ATE 1

phosphorylation of Elk-1 element in MCF-7 cells through MAPK-dependent I strogen receptor-mediated activation of the serum response

Duan R. Nie W. Burghardt R C: Safe S

CONTRACT NUMBER CORPORATE SOURCE A & M University, College Station, Texas 77843-4466 USA Department of Vetermary Physiology and Pharmacology, Texas F809106 (NIFHS)

1:S09253 (NIFIIS)

11500-8 JOTRNAL OF BIOLOGICAL CHEMISTRY, (2001 Apr 13) 276 (15)

PUB COUNTRY Journal code/ HIV: 2985121R/ISSN: 0021-9258 United States

Journal: Article, (JOURNAI, ARTICLE)

LANGUAGE FILE SEGMENT English Priority Journals

INTRY MONTH 200105

INIKY DATE Last Updated on STN: 20010529 Fatered STN 20010529

Entered Medline, 20010524

VB 17beta-1 stradiol (F2) induces e-fos protooneogene expression in MCF-7 human breast cancer cells, and deletion analysis of the e-fos promoter

> of the MAPK pathway and increased binding of the serum response factor and negative Elk-1, wild type, and variant GAL4addition, ERalpha-negative MDA-MB-231 breast cancer and Chinese hunster transforming growth activation of an SRF by F2. Both F2 (FRalpha-dependent) and growth factors Elk-1 to the SRE. Sul ovary cells were used mitogen-activated protein kinase (MAPK) pathway activation by E2, and determined by mutational analysis of the promoter, analysis of receptor for TGF-alpha, only hormone-induced activation was observed in Elk-1 is an important downstream target associated with Elk-1 at scrines 383 and 389 in the C-terminal region of transcriptional activation of the SRE by E2 was due to ERalpha activation (ERalpha)-dependent activation of gene expression through the SRE was E2-responsive. The mechanism of ligand-activated estrogen receptor alpha showed that the serum response element (SRE) at -325 to -296 was Ras MAP & pathway: (FRalpha-independent) activated the SRF in breast cancer cells via the Elk-1 fision proteins confirmed that phosphory lation of bsequent studies with dominant factor alpha (TGF-alpha) as a positive control. In however, in ER-negative CHO cells that do not express a as reference cell lines. The results showed that

cells transfected with ERalpha.

(FILE HOME ENTERED AT 09/29/23 ON 15 MAY 2002)

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E17 VEGF RECEPTOR (HUMAN GENE FLT-4) CN

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1.19 . ANSWER 1 OF 2 REGISTRY COPYRIGHT 2002 ACS

252974-32-6 REGISTRY

Vaset lar endothelial growth factor (human 148-amino acid isoform

precursor) (9CI) (CA INDEX NAME)

OTHER NAMES: GenBank AF091352-derived protein GI 5901561

VEGF (human 148-amino acid isoform)

PROTEIN SEQUENCE

47.1 TOS

SEQ 1 MNFLLSWVIIW SLALLLYLIIII AKW SQAAPMA EGGGQNIIIIEN VKEMDVA QRS

151 KIII EVQDPQT CKCSCKNTDS RCKM 51 YOHPIFTI VID JEQEYPDEIE YIEKPSCVPL MRCGGCCNDE GLECVPTEES 101 NITMOIMRIK PHQGQHIGEM SELQHNKCEC RPKKDRARQE N°CGPCSERR Unspecified

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2 REFERENCES IN EILE CAPLUS (1967 TO DATE)
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THE REGISTRY ENTERED AT 09:29:35 ON 15 MAY 2002

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1 °C 2738 S 142805-58-1 RN

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<u>-</u> 108 S ETS-LIKE?(S)TRANSCRIPTION

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6883 S G.M.4 OR G.M.!4

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<u>-</u> 11 S 1 10(S)[11

26 S 1.14 OR 1.15

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FILE INFIDIANE, CAPILIS, BIOSISTENTERED AT 09:53:36 ON 15 ALAY 2002

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FULL ESTIMATED COST

SESSION 14.67

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE 1.0.1.1

ENTRY SESSION

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J.20 s vegf or (vascular endothelial growth factor)
 2281: VEGF OR (VASCULAR ENDOTHELIAL GROWTH FACTOR)

· s 120(p)17

295 L20(P) L7

s 120(p)18

8 I 20(P) I.8

s 120(F)198

of L-numbers, enter DISPLAY HISTORY at an arrow prompt (The L-number entered could not be found. To see the definition GN. 104. J.O. 86.

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d ti 122 1-8

1.22 ANSWER LOFS MEDI INE

Ti Acide extracellular pH induces vascular endothelial growth factor (VEOF) of low pll-induced VEGE in Eurrar glioblastoma cells via ERK1/2 MAPK signaling pathway; mechanism

L22 ANSWER 2 OF 8 MEDILLE

receptors and activates mitogen-activated protein (MAP) kinase of dog retinal capillary endothelial cells. Vascular endothelial growth factor (VEGF) enhances the expression of

1 22 ANSWER 3 OF 8 MEDIANE

[11] Focal adhesion kinase, Rap1, and transcriptional induction of vascular endothelial growth factor.

- 1.22 ANSWER 4.01 & BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
- 11 Vascular endothelial growth factor (VEGF) enhances the expression of retinal capillary endothelial cells receptors and activates initogen-activated protein (MAP) kinase of dog
- 1.22 ANSWER 5 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
- 11 Focal adhesion kinase. Rap1, and transcriptional induction of vascular endothelial growth factor.
- ANSWER 6 OF 8 CAPITUS COPYRIGHT 2002 ACS
- of low ph-induced VEGF in human ghoblastoma cells via ERK1/2 MAPK signaling pathway. Mechanism Nordre extracellular pH induces vascular endothelial growth factor (VEGF)
- ANSWER 7 OF 8 CAPITUS COPYRIGHT 2002 ACS
- receptors and activates mitogen-activated protein (MAP) kinase of dog retinal capillary endothelial cells Vascular endothelial growth factor (VEGF) enhances the expression of
- 122 ANSWER & OF & CAPILUS COPYRIGHT 2002 ACS
- 11 Local adhesion kmase. Rapt. and transcriptional induction of vascular endothehal growth factor

PROCESSING COMPLETED FOR L22 dup rein 122

3 DUP REM L22 (5 DUPLICATES REMOVED)

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ANSWER LOFF MILICIAN

DUPLICATE I

- 11 Neidic extracellular pH induces vascular endothelial growth factor (VEGF) of low pH-induced VEGF in human ghoblastoma cells via FRK1/2 MAPK signaling pathway (mechanisn
- TES VASWER DOFF MEDIAN

DUPLICATE 2

- 11 Focal adhesion kinase, Rap1, and transcriptional induction of vascular
- ANSWIR 3 OF 3 MEDIANE DUPLICATE 3

11 Vascular endothelial growth factor (VEGF) enhances the expression of

receptors and activates mitogen-activated protein (MAP) kinase of dog

retinal capillary endothelial cells

endothelial growth factor.

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ACCESSION NUMBER 2001043622 MEDIANE DOCUMENT NUMBER 20431646 PubMed ID: 10977134 125 ANSWIR FOF 3 protein (MAP) kinase of dog retinal capillary endothelial expression of receptors and activates mitogen-activated Vascular endothelial growth factor (VEGF) enhances the MEDLINE DUPLICATE 3

Murata M; Kador P F: Sato S

CORPORATE SOURCE: Laboratory of Ocular Therapeutics, National Eye Institute,

Nitional Institutes of Health, Bethesda, Maryland

20892-1850, USA.

SOLECT: JOURNAL OF OCUT AR PHARMACOLOGY AND THERAPEUTICS, (200)

lo (4) 383-91.

Journal code: CBR, ISSN: 1080-7683.

PUB. COUNTRY: United States

Journal, Article; (JOURNAL ARTICLE)

FILESEGVIENT LANGU AGE English Priority Journals

ENTRYMONTH 200012

ENTRY DATE Entered STN: 20010322

Last Updated on STN: 20010322

Entered Medline: 20001207

AB. Since the galactose-fed dog is an animal model that develops the advanced stage of proliferative retinopathy, the effects of vascular

endothelial growth factor (VEGF) on

were investigated. Dog retinal endothelial cells were cultured at 37 protein (N/AP) kinase pathway of dog retinal capillary endothelial cells cell grewth, receptor expression and the activation of mitogen-activated

expression was examined by RT-PCR, and activation of MAP kinase was with et do helial cell growth factor (ECGF). VEGF receptor degrees C under 5% carbon dioxide atmosphere in CS-C medium supplemented

examired with antibody against phospho-Elk-1 (Ser383). dog endothelial cells When growth factors were removed from the culture medium, cell survival of was significantly reduced. Addition of VEGF

protected these cells from cell death induced by growth factor starvation VEGF also enhanced tube formation in dog endothelial cells and

increased the expression of two VEGF receptors, FR-I and

KDR Flk-1. Cells treated with VEGF also displayed the

phosphorelation of the transcription factor, EIK-1

Addition of the tyrosine kinase inhibitor, genistem, eliminated

phosphorylation. These data confirm that cell growth and tube formation of VEGIT-induced cell growth and Elk-1

dog retinal capillary endothelial cells are stimulated by VEGF

Flt-1, and activates the p44-42 MAP kmase pathway **VEGF** also increases the expression of the receptors, KDR and

d ibih ab 2

1.25 ANSWER 2 OF 3 VIEDLINE. DUPLICATE 2

ACCESSION NUMBER 2000429884 MEDIANE

DOCUMENT NUMBER

20341838 PubMed ID: 10880549

Focal adhesion kinase, Rap1, and transcriptional induction

COMMENT ACITION: of vascular endothelial growth factor. Sheta F. V, Harding M. A; Conaway M.R. Theodorescu D. Comment in: J Natl Cancer Inst. 2000 Jul 5,92(13):1030-1

CORPORATE SOURCE University of Virginia Health Sciences Center. Department of Molecular Physiology and Biological Physics.

Charlottesville 22908, USA.

CONTRACTATABER 11732DK0T166-01 (NIDDK)

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                                                                                                                                                                                                                                                                                                                                      (FILE THOME FINTERED AT 09:29:23 ON 15 MAY 2002)
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              transformation-dependent, mechanism for stimulus-specific regulation of
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          transcription via LAK and Rap1 provides a novel Ras-independent, but
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         of convergent MAPK-signaling pathways arising from different upstream
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        in malignant (P=0001) but not in nonmalignant (P=37) prostate cells.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                      the cell-cell contact process, and reporter activity was assessed at
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        construct and with constructs that express dominant negative or activated
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       cells were also cotransfected with a VEGF promoter-reporter
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                              factor, can be induced by cell-cell contact. In the current work, we
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    functions. Transcription of vascular endothelial
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 nucleus, informing decisions on growth, angiogenesis, and other cell
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                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   tumor VEGF expression via MAPK.
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    extracellular stimuli CONCI USIONS: Cell contact induction of VEGF
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           contact suggests that Rapl is a key factor in regulating the specificity
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           Ras-sensitive EIK-1 chimeric reporter by cell-cell
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             was Ras independent. In addition, transcriptional activation of a
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             it required the activity of focal adhesion kinase (FAK), Rap1, and Raf and
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           This process was mediated via a mitogen-activated protein kinase (MAPK):
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                           transcriptional activation of a VEGF promoter-reporter construct
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                          cell-cell contact, but not extracellular matrix components, resulted in
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                               various cell densities. All P values are two-sided, RESCLTS; Direct
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        versions of signal transduction proteins hypothesized to be involved in
                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                       cell contact on expression of VEGF messenger RNA Transformed
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CANCERLIT, CAPIL'S, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPC, ODFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...'

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II Acadic extracellular pH induces vascular endothelial growth factor (VEGF) in human ghoblastoma cells via FRK1-2 MAPK signaling pathway - Mecharism of low pH-induced VEGF

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11 Focal adhesion kinase, Rap1, and transcriptional induction of vascular endothehal growth factor

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11 Vascular endothelial growth factor (VEGF) enhances the expression of receptors and activates mitogen-activated protein (MAP) kinase of dog retinal capillary endothelial cells

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- CODEN: PINND2

33 ANSWER 49 OF 50 CAPIUS COPYRIGHT 2002 ACS

II Araporter gene system for identifying morphogen analogs that activate the osteogenic protein-1-responsive element

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LOG ANSWER 50 OF 50 CAPILI'S COPYRIGHT 2002 ACS

SO PCT Int Appl. 58 op

CODEN PINND2

11 Reporter gene methods for identification of compounds that modulate

SO [U.S., 93 pp]. Cont-in-part of U.S. Ser. No. 555,196, abandoned

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ANSWER LOFS

- 11 Epidermal growth factor receptor transcriptionally up-regulates vascular endothehal growth factor expression in human glioblastoma cells via a induced by hypoxia. pathway involving phosphatidylinositol 3'-kinase and distinct from that
- SO CANCER RESEARCH (2000 Oct 15) 60 (20) 5879-86.
 Journal code: CNF ISSN: 0008-5472.

L35 ANSWER 2 OF 5 MEDI INF

- 31 Growth factor activation of the estrogen receptor in vascular cells occurs
- so Journal of CHNICALINYESTIGATION (1998 Jun 15) 101 (12-7851-61 Journal code 1187, 7802877 ISSN 0021-9738.

LAS ANSWER 3 OF 5 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC

- [1] Epidermal growth factor receptor transcriptionally up-regulates vascular induced by hypoxia pathway involving phosphatidylinositol 3'-kinase and distinct from that endothelad growth factor expression in human glioblastoma cells via a
- SO: Cancer Research, (October 15, 2000) Vol. 60, No. 20, pp. 5879-5886, print ISSN 0008-5472

FAS ANSWER 4 OF 5 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

11. Growth factor activation of the estrogen receptor in vascular cell soccurs via a mitogen-activated protein kinase-independent pathway

> SO Journal of Clinical ISSN 0021-9738. Investigation, (June 15, 1998) Vol. 101, No. 12, pp.

LIS ANSWER SOFS CAPIL'S COPYRIGHT 2002 ACS

- The Epider nat growth factor receptor transcriptionally up-regulates vascular endothelial growth factor expression in human glioblastoma cells via a induced by hypoxia pathway involving phosphatidylinositol 3'-kinase and distinct from that
- SO Cancer Fesearch (2000), 60(20), 5879-5886 CODEN: CNRLAS: ISSN: 0008-5472

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L35 ANSWER 4 OF 5 ACCESSION NUMBER: BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC 1998:357915 BIOSIS

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Finase-independent pathway. vascular cell soccurs via a mitogen-activated protein Growth (factor activation of the estrogen receptor in

NUTIOR(S E.; Baur, Wendy E.; Mendelsohn, Michael F. Karas, Richard H. (1); Gauer, Elizabeth Ann; Bieber, Halbe

CORPORATE SOURCE 'N'ashington St. No. 80, Boston, MA 02111 USA (1) Mol. Cardiol. Res. Cent., New Engl. Med. Cent., 750

Journal of Clinical Investigation. (June 15, 1998) Vol.

101, No. 12, pp. 2851-2861. SSN: 0021-9738.

DOCUMENT TYPE . Article

LANGUAG :: English

All. The classical estrogen receptor ERalpha mediates many of the known cardiovascular effects of estrogen and is expressed in male and female estrogen response element-based, and vascular endothehal cells (PV occur in cells from reproductive tissues, but has not been investigated vascular cells. Estrogen-independent activation of ERalpha is known to in human saphenous previously in vascular cells. In this study, transient transfection assays EC) demonstrated FRalpha-dependent activation of vein smooth muscle cells (HSVSMC) and pulmonary vein

endothelial growth **factor**-based

via m togen-activated protein (MAP) kinase induced In nonvascular cells, reporter plasmids by ERalpha-mediated gene expression can be activated both estrogen-deficient FBS (ED-F3S) and EGF

found that pharmacologic inhibition of NINP kinase did phosphorylation of serine 118 of ERalpha. However, in vascular cells, we

conta ning an alanine not alter EGF-mediated ERalpha activation. In addition, a mutant EE in both HSVSMC and PVEC. Furthermore, constitutively active MAP activated to the same degree as the wild-type receptor by FD-FBS and FGF -for-serine substitution at position 118 was

also stimulate ERalpha-mediated gene expression in vascular cells, but but MAPKK inhibited ER activation in PVFC. We conclude that growth factors pathway distinct from that reported previously in nonvascular cells find that this occurs via a NLAP kinase-independent kinase kinase (MAPKK) activated ERalpha in Cost cells as expected.

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139 ANSWER LOF 44 MEDIANE

- of low pH-induced VFGF Acidic extracellular pH induces vascular endothelial growth factor (VEGF) in human ghoblastoma cells via ERK1-2 MAPK signaling pathway: mecharism
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2002 Mar 29) 277 (13) 11368-74 Journal code 2985121R ISSN 0021-9258

L 39 L NSWER 2 OF 44 VII DL IV DUPLICATE I

- 11. I ithium induces gene expression through lymphoid enhancer-binding factor '1-cell factor responsive element in rat PC12 cells.
- SO | NEUROSCIENCE LETTERS, (2002 Jan 4) 317 (1) 50-2 Journal code 7600130 ISSN: 0304-3940.

L39 ANSWER 3 OF 44 AIL DI INE

DUPLICATE 2

- (II) Presentlin I regulates beta-catenin-mediated transcription in a glycogen synthase kinase-3-independent fashion.
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 Oct 19) 276 (42) 38563-9 Journal code 2985121R ISSN 0021-9258

139 ANSWER 4 OF 14 MEDITAL

DUPLICATE 3

- 11 Induction-independent recruitment of CREB-binding protein to the e-fos serum response element through interactions between the bromodomain and
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 Feb 16) 276 (7) \$213-21 Journal code 1IIV 2985121R ISSN: 0021-9258

THE ANSWER SOF 44 MEDIANE

- co-repressors All TelHMO box transcription factors interact with Groucho-related
- SO NUCLEIC ACIDS RESEARCH (2001 Apr 1) 29 (7) 1410-9 Journal code O8I J04H-011 ISSN 1362-4962

139 ANSWER 6 OF 44 MEDILINE

- beta-catenin-LEF-1 to activate matrily sin transcription in intestinal The PLA3 subfamily of Ets transcription factors synergizes with
- SO MOLECULAR AND CELLULAR BIOLOGY, (2001 Feb) 21 (4) 1370-83. Journal code NGY, 8109087 ISSN 0270-7306

1.39 NSWFR 7 OF 44 AFDI INT

DUPLICATE 5

- TI Reduced expression of Wnt-1 and E-cadherin, and diminished beta-catenin C-alpha stability in MCF-7 breast cancer cells that overexpress protein kinase
- SO INTERNATIONAL Journal code: 9306042 ISSN: 1019-6439. JOURNAL OF ONCOLOGY, (2001 Dec) 19 (c) 1227-33

1.39 ANSWER 8 OF 44 MEDIANE

9 4.I.V. OF Id. 1C

- 11 Tumor suppressor PTEN inhibits nuclear accumulation of beta-catenin and T cell lymphoid enhancer factor 1-mediated transcriptional act vation.
- SO JOUENAL OF CELL BIOLOGY, (2001 Jun 11) 153 (6) 1161-74. Journal code: 0375356 JSSN: 0021-9525.

L39 ANSWTR 9 OF 44 MEDI INE

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- TI The anthocyanidins eyanidin and delphinidin are potent ir hibitors of the epidermal growth-factor receptor.
- SO JOURNAL OF AGRICULTURAL AND FOOD CHEMISTRY, (2007 Feb) 49 (2) 958-62 Journal ec de: H3N: 0374755, ISSN: 0021-8561

139 ANSWER 10 OF 44 AUTULINE DITPLICATE 8

- TI Characterization of the human UDP-galactose:ceramide galactosyltransferase gene promoter.
- SO BIOCHIMICA ET BIOPHYSICA ACTA (2001 Feb 16) 1517 (3) 416-23. Journal code: A0W; 0217513 ISSN: 0006-3002.

1.39 ANSWER 11 OF 44 MEDIANE

- The Perturbation of the tight junction permeability barrier by occludin loop. pepfides activates beta-catenin TCF LEF-mediated transcription
- SO EMBO Rep. (2001 Journal ender DOT: 100963049 [SSN: 1469-221N Apr) 2 (4) 306-12.
- 139 ANSWER 12 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICM, ABSTRACTS INC.
- TI ITE11 inds to the promoter of neutrophil clastase gene to up-regulate its expression.
- SO Blood (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 282a-283a http://www.bloodjournal.org/print. Part I Orlando, Florida, USA December 07-11, 2001 Meeting Info.: 43rd Annual Meeting of the American Society of Hernatology.

139 ANSWER 13 OF a <u>+</u> MEDIANE

ISSN 0006-4971.

- 11 Mutant E-cadherin breast cancer cells do not display constitutive Wnt Sur rugis
- SO CANCER RESEARCH, (2001 Jan 1) 61 (1) 278-84 Journal code: CNF ISSN: 0008-5472.

1.39 . N.S.V.ER 14 OF 44 MEDIANE

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- II Insulin and IOF-1 stimulate the beta-catenin pathway through two signallir g cascades involving GSK-3beta inhibition and Ras activation.
- SO ON COGENE, (2001 Jan 11) 20 (2) 252-9 Journal code: ONC; 8711562, ISSN: 0950-9232

139 ANSWER IS OF 44 AIFIDI INI

DUBLICATE TO

- 11 Protein kinase C mu selectively activates the mitogen-activated protein kmase (MAPK) p42 pathway.
- SO TEBS LETTERS, (2001 Mar 9) 492 (1-2) 39-44 Journal code ECH, 0155157 ISSN 0014-5793

L 19 ANSWER TO OF 44 MEDITINE

- Wnt pathways Transcriptional regulation by Smads; crosstalk between the TGP-beta and
- SO JOURNAL OF BONE AND IOINT SURGERY AMERICAN VOLUME (2001) 83-A Suppl
- (Pt 1) S31-9 Ret 40
- Journal code: HJR, (014030-ISSN-0021-9355

ANSWER 17 OF 14 MEDI INE DUBLICATE II

- 11 A bombesin receptor subtype-3 peptide increases nuclear oncogene
- expression in a MEK-1 dependent manner in human lung cancer cells.

 SO EUROPEAN IOURNAL OF PHARMACOLOGY (2001 Jan 19) 412 (1) 13-20 Journal code 1254354 ISSN: 0014-2999

1 39 ANSWER 18 OF 44

- without activating the ERK, JNK, p38 MAPK or PI3K. Akt pathways Rit, a non-lipid-modified Ras-related protein, transforms NIH3T3 cells DUPLICATE 12
- SO ONCOGENE (2000 Sep 28) 19 (41) 4685-94 Journal code ONC ISSN 0950-9232

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- [11] Increased effect of interferon gamma on PDGF-induced c-tos gene transcription in glomerular mesangial cells: differential effect of the transcriptional coactivator CBP on STATTalpha activation.
- SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2000 Jul 14)
- Journal code, 9Y8, 0372516, ISSN 0006-291N

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- 11 Characterization of hPRP4 kinase activation; potential role in signaling.
- SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS (2000 May 10) [1]
- Journal code (372516, ISSN 0006-291N

139 ANSWER 21 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

- Transcriptional regulation and apoptosis induction by Tef beta-catenin complex in various T-cells.
- SO: Korean Journal of Biological Sciences, (December, 2000) Vol. 4, No. 4, pp 1888 1226-5071 189-39-4 print

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- SO JOURNAI OF CELL BIOLOGY (2000 Apr 17) 149 (2) 249-54 Journal code HMV (0375356 ISSN: 0021-9525

L39 ANSWE 23 OF 44 MEDILINE

- PUBLICATIVITA
- 11 Atypical protein kinase C-zeta stimulates thyrotropin-independent proliferation in rat thyroid cells.
- SO ENDOCRINOLOG lournal code: FGZ; 0375040 [ISSN: 0013-7227 Y. (2000 Jan) 141 (1) 146-52

1.39 ANSWER 24 OF 44 MEDIANE

- TI | Kat IEH receptor type 2 exhibits higher basal signaling activity than TRH receptor type 1.
- SO FNDOCRINOLOGY (1999 Oct) 140 (10) 4916-9 Journal code FGZ (0375040 ISSN: 0013-7227

1.39 ANSWI R 25 OF 44 MEDITINE

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- TI Regulation of MCL family to the induction of hematoporetic cell differentiation. mechanism links expression of a viability-promoting member of the BC...2 I through a serum response factor EIk-1-mediated
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Jan. 5) 274 (3) 1801-13 Journal code: HIV; 2985121R JSSN: 0021-9258

1.39 ANSWER 26 OF 44 MEDILINE

- DUPLICATE 16
- e-fos serum response Nove roles of specific isoforms of protein kmase C in activation of the element.
- SO VIOLECLE AR AN Journal code: 8109087 JSSN: 0270-7306. D CFI I ULAR BIOLOGY, (1999 E-b) 19 (2) 13/3-24

1.39 ANSWER 27 OF 44 MEDILLE

- DUPLICATE 17
- 11 Protein phosphatase protein expression. 2Δ suppresses MAP kinase signalling and ectopic
- SO CELLULAR SIGN Journal ender AVB 8904683 ISSN 0898-6568 MTING, (1999, Nug) 11 (8) 575-80

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- II Nitric oxide regulates shear stress-induced early growth (esponse-1) endothel al cells. Expression via the extracellular signal-regulated kinase pathway in
- SO | CIRCULATION RESEARCH (1999 Aug 6) 85 (3) 238-46 Journal code: DAJ: 0047103 JSSN: 0009-7330.

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- II The C-terminal transactivation domain of beta-catenin is necessary and sufficient for signaling by the LEF-1 beta-catenin complex in Nenopus
- SO MECHANISMS OF DEVELOPMENT, (1999 Mar) 81 (1-2) 65-74 Journal code: ANF: 9101218 ISSN: 0925-4773.

1.39 ANSWER 30 OF a MEDITAL

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- 11 Direct regulation of the Xenopus engrailed-2 promoter by the Wint signaling for Wint signaling during neural patterning in Nenopus. pathway, and a molecular screen for Wint-responsive genes, confirm a role
- SO MECHANISMS OF DEVELOPMENT (1999 Sep) 87 (1-2) 21-32 Journal code: ANF, 9101218 ISSN: 0925-4773.
- L39 ANSWER 31 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC Wrt signaling and transcriptional control of Siamois in Nenopus embryos.

SO. Proceedings of the National Academy of Sciences of the United States of America, (May 12, 1998) Vol. 95, No. 10, pp. 5626-5631.

L39 ANSWER 32 OF 44 ALL DI IN

- during embry ogenesis in the mouse I wo members of the Tef family implicated in Wint beta-catenin signaling
- SO MOLECULAR AND CHILLIAR BIOLOGY, (1998 Mar) 18 (3) 1248-50 Journal code, NGY, 8109087, ISSN, 0270-7306.

THE MINSWITE BY OF 44 7:1:DI | 7:1

- The CRFB-binding protein (CBP) cooperates with the serum response factor
- for transactivation of the c-fos serum response element. SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Dec 5) 272 (49) 310-6-21. Journal code HIV, 2985121R ISSN 0021-9258.

TWINDHINE PERSONAL MEDITINE

- 11. Gastrin and phorbol 12-invristate 13-acetate regulate the human histidine decarboxylase promoter through Raf-dependent activation of extracellular signal regulated kinase related signaling pathways in gastric cancer
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Oct 24) 272 (43) 27015-24 Journal code HIV, 2985121R ISSN: 0021-9258.

1 30 NNSW TR 35 OF 44 DUPLICATE 20

- 11 Growth hormone regulates ternary complex factors and serum response factor
- absociated with the c-fos serum response element. SO: JOURNAL OF BIOLOGIC M. CHEMISTRY, (1997 Oct 10) 272 (41) 25951-8 Journal code 1111/, 2985121R 188N 0021-9258

ANSWER ROOF 44 NIT DI INT DUPLICATE 21

- 11 Identification of a replication-competent pathogenic human immunodeficiency sirus type I with a duplication in the TCF-1alpha region
- but lacking NF-kappaB binding sites SO JOURNAL OF VIROLOGY, (1997 Feb) 71 (2) 1651-6 Journal code | KCV | 0113724 | ISSN | 0022-538N

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- DUPLICATE 22
- TI Functional role of extracellular signal-regulated protein kinases in gastric acid secretion.
- Journal code 3U8, 0370511 ISSN 0002-9513. AMERICAN TOTRNA, OF PHYSIOLOGY (1997 Dec) 273 (6 Pt 1) 611263-72

VNSWTR 38 OF 44 MINDINE

- activation of the serum response element and in cell transformation Viole for the small GTPase Rac in polyomavirus middle-T antigen-mediated
- SO ONCOCENE (1997 Mar 13) 14 (10) 1235-41 Journal code ONC; 8711562 ISSN 0950-9232

TREAL OF BELLEVIEW OF THE MIDINE

DUPLICATE 24

- The Molecular mechanisms for the growth factor action of gastrin. SO EXMERICAN JOURNAL OF PHYSIOLOGY, (1997 Oct. 273 (4 Pt.1) 6891-8. Journal code 3U8 0370511 ISSN 0002-9513

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- TI Molecular mechanisms for somatostatin inhibition of c-fos gene expression
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1.39 ANSWER 41 OF 44 MEDLINE DUPLICATE 26

11 Urea-inducible Egr-1 transcription in renal inner medullary collecting activation. duct (m. MCD3) cells is mediated by extracellular signal-regulated kinase

STATES OF SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED

Journal code: PV3; 7505876, ISSN: 0027-8424. VMFRICA (1996 Oct 1) 93 (20) 11242-7.

1.39 ANSWER 42 OF 44 MEDILINE

DUPLICATIF 27

II Regulation of mitogen-activated protein kinases by a calcium calmodulin-

SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED dependent protein kinase cascade.

Journal code: PV3; 7505876, ISSN: 0027-8424. MIFRIC V (1996 Oct 1) 93 (20) 10803-8. STATES OF

1.39 .XNSWER 43 OF 44 MEDIANE

DUPLICATE 28

11 Serun response element and flanking sequences mediate the synergistic acetate and cholera toxin in AKR-2B cells. transer ptional activation of e-fos by 12-O-tetradecanoylphorbol-13-

SO CELLOROWIH AND DIFFERENTIATION, (1995 Aug) 6 (8) 955-64 Journal code: AVII: 9100024 JSSN: 1044-9523.

1.39 ANSW TR 44 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL A STRACTS INC

- TI Human immunodeficiency virus I tat stimulates transcription of the transferming growth factor alpha gene in an epidermal growth
- SO Cell Growth & Differentiation, (1994) Vol. 5, No. 1, pp. 87-93

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Elk-1 alpha-Synuclein forms a complex with transcription factor

CORPOR VIT SOURCE NOTITIOR Iwata A: Miura S: Kanazawa I: Sawada M: Nukina N

SOURCE: Wako-shi, Saitama, Japan. IF SOURCE: I aboratory for CAG Repeat Diseases, Molecular Neuropathology Group, RIKEN Brain Science Institute.

Journal code: JAV: 2985190R. ISSN: 0022-3042. JOURNAL OF NEUROCHEMISTRY, (2001 Apr.) 77 (1) 239-52

P' 13. COL'NTRY: Journal, Article; (JOURNAI, ARTICLE) United States

LANG JAGES English

FILL SECVIENT Priority Journals

ENTRY MONTH 200105

ENTRY DATE: Last Updated on STN: 20010517 Entered STN: 20010517

Entered Medline: 20010503

VB alpha-Synuclem has been identified as a component of Lewy bodies in alpha-symuclein in the pathogenesis, we searched for molecules interacting inclusions (GCIs) in multiple system atrophy (MSA). To explore the role of Parkinson's disease and diffuse Lewy body disease, and ghal cytoplasmic with alpha-synuclein and discovered that GCIs are stained by anti-

Elk-1 antibody. To seek the role of Elk-

I in synucleimopathies, we cottansfected alpha-synuclein and

EIk-1 to cultured cells, and found small granular

alpha-synuclein and Elk-1 were co-immunoprecipitated structure complexes where the two molecules colocalized. Moreover

FTS and B-box domains of Elk-1 was required. Although from the cell lysates. For formation of the complex, the presence of both

there was no evidence of direct binding between alpha-synuclein and

Elk-1, we discovered that alpha-synuclein and

FIk-1 both bind to FRK-2, a MAP kinase. The effect of

alpha-symuclem on the MAP kinase pathway was assessed using the

Pathdetect system, which showed prominent attenuation of

Elk-1 phosphorylation with alpha-synuclein, and

especially ASST mutant. Our results suggest that alpha-synuclein reacts oligodendrocytes and lead to neurodegeneration in Parkinson's disease and with the MAP kinase pathway, which might cause dystunction of neurons and

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DOCUMENT NUMBER 131 143353

converge to MFK1 FRK2 and activate the Elk1 occurs via multiple signal transduction pathways that Activation of the 91.3 eCAF chemokine by phorbol esters

VI THOR(S) transcription factor Martins-Green, Manuela Li, QiJing: Vaingankar, Sucheta M., Green, Harry M.;

CORPOR VIT SOURCE Riverside, C.A. 92521, USA Department of Biology, University of Californa.

Journal of Biological Chemistry (1999), 274(22).

50451-15405

CODI N. JBCHA3, ISSN: 0021-9258

PU 131 15111 K Biology American Society for Biochemistry and Molecular

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AB—Using primary fibroblasts in culture, the authors investigated the signal these tumor promoters), (2) a contribution involving tyrosine kinases, and promoters, activate the 91-3 gene and its chemokine product the chicken dexamethasone. All 3 of these pathways converge into the (3) a larger contribution via pathways that can be interrupted by by phorbol 12.13-dibutyrate (PDBu) via 3 pathways: (1) a small transduction mechanisms by which phorbol esters, a class of tumor system, the authors show that although both the AP-1 and PDRILkappa. H (a mitogen-activated protein kinases, MEKLERK2. Using a luciterase reporter contribution via protein kinase C (the commonly recognized pathway for chemotactic and angiogenic factor (cCAF). This gene is highly stimulated N1 kappa B-hke factor in chickens) response elements are capable of

> expression. Electrophoretic mobility shift assays and functional studies activation in these normal cells, regions of the 9E3 promoter contg. them are unresponsive to PDBu stimulation. In contrast, the authors show for phorbol esters leads to activation of the Elk1 transcription factor, which using PathDetect systems show that stimulation of the cells by the first time that activation by PDBu occurs through a segment of the elements abrogates 91 promoter contg. Fik I response elements; deletion and mutation of these (3) chicken chemotactic and angiogenic factor

REFERENCE COUNTS binds to its element in the 9E3 promoter THERE ARE 81 CITED REFERENCES AVAILABLE FOR

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SO Placenta, (January, 2002) Vol. 23, No. 1, pp. 100-101, pr.nt.

ISSN: 0143-4004.

ANSWER 2 OF 63 CAPIA'S COPYRIGHT 2002 ACS

11 Preparation of pyrazines as modulators of vascular

endothelial growth factor (VEGF)

SO PCT Int Appl., 202 pp. receptor tyrosine kinase.

CODEN: PINND2

ANSWER 3 OF 63 CAPIL'S COPYRIGHT 2002 ACS

II Human stress genes identified using DNA microarrays

SO. U.S. Pat. Appl. Publ., 57 pp., Cont.-in-part of U.S. Ser. No. 441,920 CODEN: USXXCO

ANSWER 4 OF 63 MEDILINE DUPLIC VIII I

receptors, and angiogenic factors by eyclooxygenase-1. autocrine paracrine regulation of cyclooxygenase-2, prostaglandin e Cyclooxygenase-1 is up-regulated in cervical carcinomas

SO CANCER RESEARCH, (2002 Jan 15) 62 (2) 424-32 Journal code: 2984705R JSSN: 0008-5472

ANSWER 5 OF 63 AHIDI IVE

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II Histone deacetylase inhibitor FK228 inhibits tumor angiogenesis SO INTELNATIONAL Journal code: 0042124 JSSN: 0020-7136. JOURNAL OF CANCER, (2002 Jan 20) 97 (3) 290-6

ANSWER 6 OF 63 CAPIL'S COPYRIGHT 2002 ACS

Isoform-specific expression of hypoxia-inducible factor-1 alpha. during

SO Melecular Endocrinology (2002) 16(2), 234-243 the late stages of mouse spermiogenesis [SSX-8880 · NSS]

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DUPLICATE 3

[11] Gordodomin A, an antifungal polyether macrolide, exhibits antiangiogenic activities via inhibition of actin reorganization in endothelial cells.

SO TOURNAL OF CELLULAR PHYSIOLOGY, (2002 Jan) 190 (1) 109-16 Journal code: 0050222 ISSN: 0021-9541

ANSWER 8 OF 63 VIEDLINE

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growth factor gene expression. factor-beta pathways on human vascular endothelial Synergistic cooperation between hypoxia and transforming growth

SO JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 Oct 19) 276 (42) 38527-35 Journal code 2985121R ISSN 0021-0258

ANSWER 9 OF 63 MEDLINE

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- 11 The constitutive photomorphogenesis 9 signalosome directs vascular endothelial growth factor production in tumor
- SO CANCER RESEARCH, (2001 Dec 1) 61 (23) 8416-21 Journal code 2984705R ISSN: 0008-5472
- NNSWIR TO OF 63 イニシニア
- 11 Regulation of vascular endothelial growth

factor by the Wnt ard K-ras pathways in colonic neoplasia.

- SO CANCER RESEARCH, (2001, Aug. 15) 61 (16) 6050-4. Journal code CNF, 2984705R ISSN: 0008-5472
- ANSWER IT OF G. BIOSIS, COPYRIGHT 2002 BIOLOGICAL ABSTEACTS INC.
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Meeting Info., 69th Scientific Sessions of the American Heart Association

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The binding of vascular endothelial growth

heparin like molecule factor to its receptors is dependent on cell surface-associated

SO J Biol Chem (1992) 267(9) 6093-8 CODEN: JBCHA3; ISSN: 0021-9258

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ANSWER COF4 MEDITAL

DUPLICATE 1

- 71 Wide spectrum of antitumor activity of a neutralizing monoclonal antibody to human vascular endothelial growth factor.
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- NSWIR 2 OF a

11 Nuclear protein interactions with the human KDR flk-1 promoter in vivo. Regulation of Sp1 binding is associated with cell type-specific DUPLICATE 2

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ACCESSION NUMBER: 97236794 ANSWER 2 OF 4 MEDITAL MEDITAL DUPLICATE 2

DOCUMENT NUMBER 07236794 PubMed ID: 9079666

with cell type-specific expression promoter in vivo. Regulation of Sp1 binding is associated Nuclear protein interactions with the human KDR flk-l

> CORPORATE SOURCE TL SOURCE: Division of Cardiology, University of Texas Medical Branch. Galveston, Texas 77555-1064, USA., camp a card clogy utmb edu Patterson C; Wu Y; Lee M F: DeVault J D: Runge M S, Haber E

CONTRACT NUMBER GM16771-02 (NIGMS)

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JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Mar 28) 272 (13)

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Journal code: HIV: 2985121R. ISSN: 0021-9258.

PUB COUNTRY: United States

Journal: Article; (JOURNAL ARTICLE)

LINGUNGE English

THE SEGMENT: ENTRY MONTH Priority Journals 199705

ENTRY DATE Entered STN: 19970514

Last Updated on STN: 20000303

Entered Medline: 19970502

VB. The encothelial cell type-specific tyrosine kinase KDR tlk-1 is a

receptor for vascular endothelial

growth factor and a critical regulator of endothelial

region. Despite the cell type specificity of KDR 4lk-1 expression, no cell pairs -110 and -25 was defined by in vitro DNase I footprinting analysis elements coordinate Sp1 binding and chromatin structure to regulate cell nucle ascmal positioning was observed. In vivo dimethylsulfate footprinting human tibroblasts and HeLa cells a pattern consistent with core promoter interactions between cell types. Protection of Sp1 binding type differences were observed in DNA-protein interactions in vitro. In proximal promoter of human KDR IIk-1. A protected sequence between base type-specific expression of KDR (lk-1). HUVEC's but not in nonendothelial cells. It is possible that distant sites was observed in HUVECs by in vivo DNase I footprinting, whereas in contrast, in vivo footprinting assays demonstrated marked differences in produced similar protection, and electrophoretic mobility, shift assays differentiation and gene regulation, we have analyzed the topology of the confirmed that DNA-protein interactions occurred within Sp1 elements in demonstrated that Sp1 was indeed the major nuclear protein binding to this in human umbilical vein endothelial cells (HUVECs). Pur fied Sp1 alone cell grow'h and development. To study mechanisms of endothelial cell

L7 ANSWER 3 OF 4 MEDLINE

94257859 MEDILINE

ACCESSION NUMBER: 94257859 PubMed ID 7515293

Vascular endothelial growth factor and its receptors.

SOL RCE. VI THOR PROGRESS IN GROWTH FACTOR RESEARCH, (1994) 5 (1) 89-97 Neufeld G Tessler S; Gitav-Goren II; Cohen T; Levi B /

Journal code: A6S; 8912757, ISSN: 0955-2235 Ref: 61

PUB COUNTRY: ENGLAND: United Kingdom

Journal: Article: (JOURNAL ARTICLE)

General Review: (REVIEW)

(REVIEW. TUTORLAL)

FILE SECMENT English Priority Journals

LANGUAGE

ENTRY MONTH 99407

ENTRY DATE Entered STN: 19940714

Entered Medline, 19940707 Last Updated on STN 20000303

VB Vascular endothelial growth factor (VEGF) is a highly specific mitogen for related to platelet derived growth factor (PDGF). It is also known as the vascular endothehal cells and an angiogenic factor that is structurally in tumour angiogenesis. Antibodies directed against VFGF can inhibit the the function of the VEGF receptors in these cells is boxine corneal endothelial cells. HeLa cells and human melanoma Several cell types which do not proliferate in response to VEGF such as growth of a variety of VEGF producing tumours. Of the various VEGF properties are transcribed from a single gene as a result of alternative permeabilization of blood vessels. Five types of VEGF mRNA encoding VEGF unclear. Recently, the tyrosine-kinase receptors encoded by the fit and cells also express cell surface VEGF receptors, but endothelial cells depends on the presence of heparin-like molecules species, the best characterized is the 168 amino acid long form (VFGF168) produced by different tumorigeme cells, and appear to play a major role meluding smooth imisele, luteal and adrenal cortex cells. VEGFs are also species which differ in their molecular mass and in their biological vascular permeability factor (VPF) because it efficiently potentiates the VEGF receptors on the cell surface of vascular splicing. VFGFs are produced and secreted by several normal cell types VF GF165 is a heparin binding growth factor, and its interaction with

1.7 ANSWER 4 OF 4 CAPI US COPYRIGHT 2002 ACS ACCESSION NUMBER 1992 208557 CAPLUS

KDR flk-1 genes were found to function as VEGF165 receptors.

DOCUMENT NUMBER 116 208557

heparin-like molecules its receptors is dependent on cell surface-associated The binding of vascular endothelial growth factor to

N THOR(S) Neufeld, Gera Gitay-Goren, Hela; Soker, Shay; Vlodavsky, Israel;

CORPORNIE SOURCE. 32000, Israel Dep. Biol., Technion, Israel Inst. Technol., Haifa.

CODEN: JBCHA3, ISSN: 0021-9258 J. Biol. Chem. (1992). 267(9), 6093-8

DOCUMENT TYPE Journal

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VB. The effect of heparin upon the interaction of vascular endothelial growth 0.1-10 mu.g ml., strongly potentiated the binding of 1251-VEGF to its indicates that 1 mu g ml. heparm induces an 8-fold increase in the conens (4 ng mL), heparm preferentially enhanced the formation of the 1251-VEGF-receptor complexes on endothelial cells. At high 1251-VEGF the dissocit const of VEOT. Crosslinking expts, showed that heparin apparent doof high affinity binding sites for VEGF, but does not affect receptors on endothelial cells. Scatchard anal. of 1251-VEGF binding factor (VEGF) with its receptors was studied. Heparin, at concis, ranging restored by the addn of exogenous heparin to the digested ceals. The 1251-VEGF binding. The binding of 1251-VEGF was completely inhibited following digestion of endothelial cells with heparinase and could be 170- and 195-kDa complexes. Premeubation of the cells with heparin strongly potentiates the formation of the 170-, 195-, and 225-kDa followed by extensive washes, produced a similar enhancement of subsequent

> enhancing effect of heparin facilitated the detection of VEGF receptors on interaction of VEGF with its cell surface receptors cell types that were not known previously to express such receptors. Evident y, cell surface-assoed, heparin-like mols, are required for the

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150977-45-0 REGISTRY

CHIERNAMIS Kinase (phosphorylating), gene flk-1 protein (9CI) (CA INDEN NAME)

Flk-1 receptor tyrosine kinase

FIK-1 KDR VEGE receptor tyrosine kinase

 \hat{Z} Gene flk-1 receptor tyrosine kinase

9 Protein kinase Flk-1

 \hat{z} VFGF receptor 'yrosine kinase 2

 $\stackrel{\cdot}{=}$ Unspecified

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5094 S VEGE(2A)RECEPTOR OR (VASCULAR ENDOTHELIAL GROWTH 63 DUP REM L3 (38 DUPLICATES REMOVED)

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L13 ANSWER LOF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC

A single autophosphorylation site on KDR Flk-1 is

essential for VEGE-2 in vascular endothelial cells. V-dependent activation of PLC-gamma and DNA synthesis

SO Exfl-O (European Vol. 20, No. 11, pp. 2768-2778. print. Molecular Biology Organization) Journal. (June 1, 2001)

[N.TO]

ISSN:)261-4189

L13 ANSWER 2 OF 7 MEDIANE

TI HIV-1-trans-activating (Tat) protein: both a target and a tool in

therapeutic approaches. SO_EIOCHEMICM, PHARMACOLOGY, (1999 Nov. 15) 58 (10) 1521-8. Ref. 89

Journal code 974, 0-01032 ISSN 0000-2952

THE ANSWER FOF 7

II Differential transcriptional regulation of the two vascular endothelial growth factor receptor genes. Flt-1, but not Flk-1

KDR, is up-regulated by hypoxia.

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THE ANSWER 4 OF 7

II Nuclear protein interactions with the human KDR flk-1 DUPLICATE I

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Journal code HIV, 2985121R ISSN 0021-9258.

11. Heterodimers of placenta growth factor vascular endothelial growth factor Endothelial activity, tumor cell expression, and high affinity binding to

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THE ANSWER 6 OF THRIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC 11 Cloning and functional analysis of the promoter for KDR flk-

I. a receptor for vascular endothelial growth factor.

SO Journal of Biological Chemistry, (1995) Vol. 270, No. 39, pp. 23111-23118 「スタス CC21-525×

1 13 11.5WFR 7 OF 7 MEDIAN

Vascular endothelial growth factor and its receptors.

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DOCUMENT NUMBER | PREV200100335431 BIOSIS THE ANSWER FOF 7 BIOSIS COPYRIGHT 2002 BIOLOGIC M. ABSTEACTS INC.

A single autophosphorylation site on KDR FIk-

Lis essential for VEGE-X-dependent activation of

PLC-gamma and DNA synthesis in vascular endothelial cells.

CORPOR VIE SOURCE (1) Department of Genetics, Institute of Medical Science, Shibuya, Masabumi (1) Takahashi, Tomoko, Yamaguchi, Sachiko; Chida, Kazuhiro;

University of Tokyo, Minato-kii, Tokyo, 108-8639.

(June 1, 2001) Vol. 20, No. 11, pp. 2768-2778, print. shibuya a imsu-tokyo ac jp Japan EMBO (curopean Molecular Biology Organization) Journal

ISSN 0261-4189

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TANGUA SCAIN ARY I AND TOTAL English Article English

> AB KDR Flk-1 tyrosine kinase, one of the two vascular phosphorylate phospholipase C-gamma and, significantly, *cduced MAP kmase phosphorylation and DNA synthesis in response to VEGF-A. Furthermore. phosphoY1175 region, we demonstrated that Y1175 is phosphorylated rapidly crucial for endothelial cell proliferation, and that this region is a new primary endothelial cells microinjected with anti-phospho Y1175 antibody properties. An in vitro kinase assay and subsequent tryptic peptide constructed a series of human KDR mutants and examined their biological mechanish s underlying the VEGF-A-induced growth signaling pathway we endotherial growth factor (VEGF) receptors, induces mitogenesis and target for anti-angiogenic reagents. findings strongly suggest that autophosphorylation of Y1175 on KDR is introduced into the endothelial cell lines by adenoviral vectors, only the differentiation of vascular endothelial cells. To understand the clearly decreased DNA synthesis compared with control celus. These Y1175 KDR, Tyr1175 to phenylalanine mutant, lost the ab lity to tyrosine in vivo in primary endothelial cells. When the mutated KDRs were mapping revealed that Y1175 and Y1214 are the two major VEGE-A-dependent natophosphorylation sites. Using an antibody highly specific to the

113 ANSWER SOF 7 MEDIANE

DOCUMENT NUMBER ACCESS ON NUMBER 96216393 96216393 PubMed ID 8621715 MEDLINE

111111 growth factor. Endothelial activity, tumor cell expression, Heterodimers of placenta growth factor vascular endothelial

and high affinity binding to Flk-1 KDR

J.A. Wang \ Cao Y; Chen H; Zhou L; Chiang M K; Anand-Apte B, Weatherbee a Fang Fa Flanagan J.G. Tsang M.L.

CORPORATE SOURCE Massachusetts 02115, USA. Department of Surgery, Harvard Medical School, Boston.

CONTRICT NUMBER PO1-CA-45548 (NCI)

SOL'ECT 3154-62. ≥. IOI NAI OF BIOLOGICAL CHEMISTRY, (1996 Feb 9) 271 (6)

Journal code/ HIV: 2985121R/ ISSN: 0021-9258

PUB. COUNTRY: United States

Journal: Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENTS Priority Journals

FAIRY MONTH ENTRY DIVING Entered STN: 19960627 199606

Entered Medline: 19960619 Last Updated on STN: 20000303

AB. Here we show that the Escherichia coli expressed monomers of placenta growth factor (Pl GF)129 and vascular endothelial growth factor (VEGF)165 can be re-folded in vitro to form PLGF VEGF heterodimers. The purified bind to this receptor cell incs. While PLOF VEOF beterodiners bind with high affinity to a PLOF YEOF heterodimers in the conditioned media of various human tumor these in vitro assays. homodimers. In contrast, PLGF129 homodimers have little or no effect in recombinant PLOF VEOF heterodimers and VEOF homodimers have potent soluble FIk-1 KDR receptor, PLGF129 homodimers fail to heterod mers display 20-50-fold less mitogenic activity than VEGF165 m to 3eric and chemotactic effects on endothelial cells. However, Pl GF VEGF We also demonstrate the presence of natural Cross-linking of 1251-ligands to human umbilical

homodimers, but not PLGF129 homodimers, form complexes with membrane the formation of PLOF VEGF heterodimers in cells producing both factors tyrosine phosphorylation of a 220-kDa protein, the expected size for the receptors. VEGF165 homodimers and PLGF VEGF heterodimers stimulate homodimers are unable to induce tyrosine phosphorylation of this protein vein endothelial cells reveals that PLOF VEOF heterodimers and VEGF165 KDR receptor in human umbilical vem endothelial cells, whereas PI GF129 These data indicate that PLGF may modulate VEGF-induced angiogenesis by

s heterologous or recombinant

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ANSWIR LOF 17 VIED IN

Antitumor activity of cytotoxic T lymphocytes engineered to target

vascular endothelial growth factor receptors.

SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STAIRS OF

Journal code 7505876 ISSN 0027-8424 MIFRICA, (2002 May 14) 99 (10) 7009-14

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DI PLICATE I

administration in adult rat brain Angrogenic and astroghal responses to vascular endothelial growth factor

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ISSN: 0006-4971.

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and accelerates resolution of experimentally induced glomerulonephritis Vascular endothelial growth factor enhances glomerular capillary repair

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SO. Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 360, print Diego, California, USA November 10-15, 2001 ISSN 0190-5295. Meeting Info: 31st Annual Meeting of the Society for Neuroscience San

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'I lumor necrosis factor downregulates vascular endothelial growth fac or

receptor Flx-1 in vivo

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11 Heterodimers of placenta growth factor vascular endothelial growth factor アデー アンマ I indothelial activity, tumor cell expression, and high affinity binding to

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angrogenesis in vivo. KDR DF-1-positive endothelial cell proliferation in vitro and Vascular endothelial growth factor-toxin conjugate specifically inhibits

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CODIN PINNO2

DUPLICATE 24

1.16 ANSWIR 37 OF 37 11 Placenta growth factor. Potentiation of vascular endothelial growth factor not to FIE-1 KDR bioactivity, in vitro and in vivo, and high affinity binding to FIt-1 but VIII DI INE

SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Oct 14) 269 (41) 25646-54 Journal code HIV, 2985121R ISSN 0021-9258

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LIG ANSWER 36 OF 37 CAPLUS COPYRIGHT 2002 ACS ACCESSION NUMBER 1994-549798 CAPLUS

DOCUMENT AUXIBER 121-149798

factor and compounds modulating their interaction FLK-1 as receptor for vascular endothelial growth

PAIENT ASSIGNER(S): フィーノー(元()) Ullrich, Axel; Risau, Werner; Millauer Birgit Max-Planck-Gesellschaft zur Foerderung der

Wissenschaften e.V., Germany PCT Int. Appl., 98 pp.

CODEN PINNI)2

FAMILY ACC NUM COUNT: 7 DOCUMENT TYPE NYGUNGH. English Patent

PATENTIN ORMATION:

PATENT NO. KIND DATE APPLICATION NO. DATE

oot 1116 O.M W.M.BG BR <u>-</u> BY, CA, CZ, FL HU, JP, KP, KR, KZ, LA, NO, NZ, PL, 10010526 WO 1993-FP3191 19931115

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96581-2661 S.1 A 19930326

WO 1993-FP3191 W 19931115

All. The present invention relates to the use of ligands for the Flk-1 receptor to the use of FLK-1 ligands, including VEGF agonists and antagonists, in expression is assoed. for the modulation of angiogenesis and vasculogenesis. The invention is and angiogenesis. the treatment of disorders, including cancer, by modulating vasculogenesis agonist or antagonist for drugs and analogs of VEOF involved in Flk-1 modulation by either that express Flk-1 and the uses of expressed Flk-1 to evaluate and screen vascular endothelial growth factor (VFCiF) as the high affinity ligand of based, ir part, on the system during vasculogenesis and angrogenesis. Engineering of host cells These results activities is described. The invention also relates indicate a major role for Fik-1 in the signaling with endothelial cells and the identification of demonstration that Fik-1 tyrosine kinase receptor

1.16 ANSWER 33 OF 37 MEDIAN DUPLICATE 21

ACCESSION NUMBER: 96216393 MEDLINE

DOCUMENT VINBER 96216393 PubMed ID 8621715

and high affinity binding to Flk-1 KDR. growth factor. Endothelial activity, tumor cell expression. Heterodimers of placenta growth factor vascular endothelial

J.A. Wang Cao Y; Fang F; Flanagan J G; Tsang M I Y; Chen II; Zhou I.; Chiang M K; Anand-Apte B; Weatherbee

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CONTRACT NUMBER: POI-CA-45548 (NCI)
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bind to this receptor. Cross-linking of 1251-ligands to human umbilical
                                                                                                 cell lines. While PI GF VEGF heterodimers bind with high affinity to a
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                                                     soluble FIK-1 KDR receptor, PI GF129 homodimers fail to
                                                                                                                                                P1 GF VEGF beterodimers in the conditioned media of various human turnor
                                                                                                                                                                                      these in vitro assays. We also demonstrate the presence of natural
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                                                                                                                                                                                                                                                                                                                                 mitogenic and chemotactic effects on endothelial cells. However, PLGF VEGF
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DOCUMENT TYPE PATENT ASSIGNER(S): DOCUMENT NUMBER 1 to ANSWER 27 OF 37 CAPITUS COPYRIGHT 2002 ACS FAMILY ACC NUM COUNTY I INVENTOR(S) PATTATINFORMATION ACCESSION NUMBER VNCH VGI PAHATAO CODEN PINNUE vascular endothelial cell surface receptor fik recombinant production, and binding by Human protein tlk-1 bp cDNA sequence. PCT Int Appl. 42 pp. 11 10070515 KIND DIVID Lyman, Stewart D. English Patent Immunex Corporation, USA 1997.440168 CAPIA'S 127/61234 WO 1996-1'S17584 19961105 APPLICATION NO. DATE

> PRIORITY APPLN INFO Al3 A pretein designated flk-1 bp binds the vascular endothelial cell surface polypeptices. Antibodies that are immunoreactive with flk-1bp are generated using the polypeptides disclosed herein. Flk-1bp competes with vectors and transformed host cells useful in producing Ilk-Ibp isolated human cDNA encoding 1lk-1bp is provided, along with expression recepto: flv-1 (fetal liver kinase 1). The nucleotide sequence of VI. 0211195 WE AUT CA, HE JP, KR, MX, NO, NZ RWEAT, BE, CH, DE, DK, ES, EL FR, GB, GR, HE TE FU MC, NL PT, SE F85215, 1-9661 O.M 19970529 122155-5001 S.1 All 1997-11162 | 19961105 19961105 19951108

is present in heart, liver, skeletal muscle, panereas, and prostate gland vascalar endothelial growth factor for binding by flk-1 and flk-1bp mRNA

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1785 S FLK-1

receptors. VEGF165 homodimers and PLGF VEGF heterodimers stimulate

homodimers, but not PLGF129 homodimers, form complexes with membrane vein endothelial cells reveals that PLGF VEGF heterodimers and VEGF165

tyrosine phosphorylation of a 220-kDa protein, the expected size for the

homodimers are urable to induce tyrosine phosphorylation of this protein.

KDR receptor in human umbiheal vein endothelial cells, whereas PLGF129

the formation of PLGF VEGF heterodimers in cells producing both factors

These data indicate that PLOF may modulate VEOF-induced angiogenes s by

1.10 E "FLK-1" CN 25 OSELK-ICY +:: v --

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1.12 9 S L1 AND L11

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1 19 ANSWER LOF 2 MEDI INE

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receptors and activates mitogen-activated protein (MAP) kinase of dog retinal capillary endothelial cells. Vascular endothelial growth factor (VEGF) enhances the expression of

L19_ANSWER 2 OF 2 MEDIANE

T1 ELK inc LERK-2 in developing kidney and microvascular endothelial assembly

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1.9 ANSWER 2 OF 2 MEDLINE

ACCESSION NUMBER 97097112 VIEDI INE

DOCUMENT VUMBER 97097112 PubMed ID: 8041020

endothelial assembly. ELK and LERK-2 in developing kidney and microvascular

Daniel T O; Stein E; Cerretti D P; St John P I; Robert B;

CORPORATE SOURCE: Abrahamson D R Division of Nephrology, Vanderbilt University Medical

CONTRACT NUMBER Center, Nashville, Tennessee, USA. DK34972 (NIDDK)

DK38517 (NIDDK)

DK47078 (NIDDK)

Journal code: KVC; 7508622, ISSN: 0098-6577 PUB. COUNTRY: United States SOL'RCE KIDNEY INTERNATIONAL SUPPLEMENT (1996 Dec) 57 873-81

Journal, Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SECRIENTS HINDY MONTH Priority Journals 199702

TATRY DATE Entered STN: 19970306

Last Updated on STN: 20000303

Entered Medline: 19970225

AB | Eph family receptor tyrosine kinases direct neuronal cell targeting. response to the exogenous ligand, LERK-2. Here we identify expression of the ELK ligand, LERK-2, in HRMEC and in primitive vascular structures of renal in crovascular endothelial cells (HRMEC), and showed that El K bundling and intercellular aggregation activity, yet their sole in identified expression of ELK (Eph-like kinase) receptors in cultured human mammalian kidney development has been unexplored to cate. We recently mediates their in vitro assembly into capillary-like structures in

renal-derived endothelial cells may distinguish LERK-2 from the angiogenic developing murine kidney. ELK and LERK-2 are expressed on endothelial human umbilical vein endothelial cell (HUVEC) responses in an in vitro Lck ligand, LERK-1 (B61), and whether endothelial cells from different and temporally coordinated in expression and may function in morphogenesis different vascular bed sources. ELK and its ligand, LERK-2, are spatially receptors (FTK and Fek) are discriminated by endothelial cells from LERK-2 Therefore responses mediated through specific Eph family capillary-like structures in response to LERK-2, but not LERK-1, under capillary-like assembly assay. HRMEC endothelial cells assembled sources may distinguish among Eph receptor ligands, we compared HRMFC and collecting ducts, glomeruli and arterioles. To explore whether uretene bud epithelium. El K and LERK-2 expression persists in mature 11k-1 antigens are also displayed on the branching that of the VI GF receptor, #k-1 FLK LERK-2 and progenitor cells of primitive microvasculature in a pattern similar to conditions that promoted HUVEC to assemble in response to LERK-1, bu-not

of the renal nucrovasculature

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120 ANSWER 1 OF 434 MILDILINE.

ACCESSION NUMBER: 2002274208 IN-PROCESS

DOCUMENT NUMBER: 22008796 PubMed ID: 12014653

The retinoid-inducible gene I, effect on apoptosis and

mitogen-activated kinase signal pathways.

NOILL I Huang Shiang-Long: Shyu Rong-Yaun: Yeh Ming-Yang: Jiang

Shun-Yuan

CORPORATE SOURCE: Graduate Institute of Life Sciences, National Defense Medical Center, Taiper, Taiwan.

ANTIC ANCER RESEARCH (2002 Mar-Apr.) 22 (2A) 709-804 Journal code: 8102988 ISSN: 0250-7005.

PUB COUNTRY Circece

Journal, Article, (JOURNAIL ARTICLE)

1.1\\Cit \(Cit\) English

FILE SEGMENT IN-FROCESS, NONINDENED; Priority Journals

INTRY DATE Entered STN 20020517

VB BACKGROUND. The retinoid-inducible gene I (RIGI), belonging to the family of type II tumor suppressor genes, was isolated from human gastric cancer was investigated in this study. MATERIALS AND METHODS: HtTA carvical and cells treated with all-trans retinoic acid. The activity of the RIGH gene Last Updated on STN 20020517

vectors that synthesized RIGI-myc or RIGI-EGFP fusion protein. Cell growth

18GH9201 gastric cancer cells were transiently transfected with express on

signal pathway trans-reporting systems. RESULTS: Expression of the respectively. CONCLUSION: The RIGH fusion proteins exhibited growth and IRTA cells transiently expressing RIG1-myc and RIG1-EGEP. activities of mitogen-activated kinase signal pathways were analyzed using kinase e-Jun N-terminal kinase and p38 mitogen-activated kinase transactivation activities of the CHOP protein was suppressed in TSG:19201 breakage. The transactivation activities of Elk1, e-Jun and CHOP negatively-regulated signal pathways of extracellular signal regulated suppressive and apoptosis-inducing activity. The protein expressing the RIG1-mye fusion protein for two days. Similarly, the proteins were suppressed by 80, 50 and 88%, respectively, in HtTA cells and RIG1-mye fusion proteins induced cellular apoptosis that was RIGH-myc fusion protein resulted in decreased cell growth. Both RIGH-EGFP Apoptosis was evaluated by the formation of in situ DNA breakage. The characterized by the presence of apoptotic bodies and in situ DNA was analyzed by measuring the incorporation of bromodeoxyuridine

5 maps of map kinase or mitogen activated protein kinase. 1—50978 MAPK OR MAP KINASE OR MITOGEN ACTIVATED PROTEIN KINASE

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L24 ANSWER LOFT MEDLINE

TI Vascular endothelial growth factor (VEGI) enhances the expression of receptors and activates mitogen-activated protein (MAP) kinase of dog retinal capillary endothelial cells.

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1.26 ANSWER I OF 34 MEDIANE

'Tl Vascular endothelial growth factor (VEGF) enhances the expression of retinal capillary endothelial cells receptors and activates mitogen-activated protein (MAP) kinase of dog

SO TOURNAL OF OCUTIAR PHARMACOLOGY AND THERAPEUTICS, (2000 Aug. 16.4)

Journal code: CBR, ISSN: 1080-7683

126 ANSWER 2 OF 34 MIDIN

- 11 De novo expression of vascular endothehal growth factor in human pancieatic cancer evidence for an autocrine mitogenic loop.
- SO GASTROENTEROLOGY, (2000 Nov.) 119 (5) 1358-72 Journal code FH3 ISSN: 0016-5085

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- TI VEGE-dependent signaling in retinal microvascular endothelial cells. SO TUKUSHIMA JOURNAL OF MEDICAL SCIENCE (1999 Dec) 45 (2, 77-91. Journal code: F91/ISSN: 0016-2590.

126 ANSWER 4 OF 34 AIF DI INE

- II IICPTPA, a protein tyrosine phosphatase that regulates vascular endothehal growth factor receptor-mediated signal transduction and biological
- SO JOURNAL OF BIOLOGIC W. CHEMISTRY, (1999 Dec 31) 274 (53) 38183-8 Journal code HIV, 2985121R, ISSN, 0021-9258

1.20 ANSWER SOFTA MEDITAL

- 11 Concomitant over-expression of vascular endothelial growth factor and i.s. receptors in panereatic cancer
- SO INTERNATIONAL JOURNAL OF CANCER (2000 Jan 1) 85 (1) 27-34 Journal code GQU, 0042124 ISSN 0020-7136

1,26 ANSWER 6 OF 34 MEDLINE

- 11 Vascular endothelial growth factor signals endothelial cell production of nitric oxide and prostacyclin through flk-1 KDR activation of c-Src SO JOURNAL OF RIOFOGICAL CHEMISTRY, (1999 Aug 27) 274 (35) 25130-5
- Journal code HIV 2985121R ISSN (021-9258)

VSW1R7OF34 MEDI INE

- 11. Vascular endothelial growth factor has neurotrophic activity and proliferation in the peripheral nervous system. sumulates axonal outgrowth, enhancing cell survival and Schwann cell
- SO JOURNAL OF NEUROSCH NCF. (1999 Jul 15) 19 (14) 5731-40 Journal code JDF 8102140 ISSN 1529-2401

1NSW1R & OF 34 MEDLINE

- myocytes transfocation of feeal adhesion kinase (p125FAK) in cultured rat cardiac Vascular endothelial growth factor induces activation and subcellular
- SO | CIRCUILATION RESEARCH (1999 May 28) 84 (10) 1194-202 Journal code | DAJ, 0047103 | ISSN: 0009-7330

126 ANSWER 9 OF 34 MEDITAL

- kinase pathway for DNA synthesis in primary endothelial cells SO ONCOGENE, (1999 Apr. 1) 18 (13) 2221-30. VFOF activates protein Emase C-dependent, but Ras-independent Raf-NFK-M vP
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activation of Ras in capillary endothelial cells. 16K human protactin inhibits vascular endothelial growth factor-induced

> SO MOLECULAR ENDOCRINOLOGY, (1999 May) 13 (5) 692-704 Journal code: NGZ; 8801431 ISSN: 0888-8809

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- II Homelogous up-regulation of KDR Flk-1 receptor expression by vascular endethelfal growth factor in vitro.
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1.26 ANSW SR 13 OF 34 MEDIANE

- TI Eleosapentaenole acid attenuates vascular endothehal growth boying carotid artery factor-induced proliferation via inhibiting Flk-1 receptor expression in endothelial cells.
- SO JOURNAL OF CE Journal code: IINB: 0050222, ISSN: 0021-9541 HTT AR PHYSIOI OGY. (1998 Aug) 176 (2) 342-9

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11 Human immunodeficiency virus tat modulates the FIk-1

KDR receptor, mitogen-activated protein

kinases, and components of focal adhesion in Kaposi's sarcoma

SO TOURNAL OF VIROLOGY, (1998 Jul) 72 (7) 6131-7 Journal code: KCV; 0113724. ISSN: 0022-538N

L26 ANSWER 15 OF 34 MEDILINE

- signa mg in mouse mesentery vascular endothelium. Vascular permeability factor vascular endothelial growth factor-mediated
- SO | CANCER RESEARCH, (1998 Mar 15) 58 (6) 1278-84 Journal code: CNF; 2984705R, ISSN: 0008-5472

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- [7] Placen'a growth factor stimulates MAP kinase and mitogenicity but not phospholipase C-gamma and migration of endothelial cells expressing Elt I.
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1.26 ANSWER 17 OF 34 MEDIANE

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- SO JOUENAL OF BIOLOGICAL CHEMISTRY, (1997 Dec 19) 272 (\$1) 32521-7 Journal code: HIV: 2985121R JSSN: 0021-0258

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- TI The 230 kDa mature form of KDR Flk-1 (VEGF receptor-2) activates the PLC-gamma pathway and partially induces mitotic signals in NHET3
- SO ONCOGENE, (1997 May 1) 14 (17) 2079-89. Journa code: ONC; 8711562, ISSN: 0950-9232

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- 11 Increase of protein tyrosine phosphorylation in ratretina after aschemia-repertusion injury
- SO INVESTIGATIVE OPHTHALMOLOGY AND VISUAL SCIENCE (1996-O.3) 37 (11) 21-10-50

Journal code, GWI, 7703701, ISSN, 0146-0404

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- cells is inhibited by the antiangrogenic factor 16-kDa N-terminal fragment of proluctin growth factor and basic fibroblast growth factor in capillary endothelial Activation of mitogen-activated protein kinases by vascular endothelial
- STAINES OF SO PROCLEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED

Journal code PV3, 7505876 ISSN: 0027-8424 AMERICA, (1995 Jul 3) 92 (14) 6374-8

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- vascular endothelral growth factor VEGF A unique signal transduction from FLT tyrosine kinase, a receptor for
- SO ONCOCH NE (1995 Ian 5) 10 (1) 135-47 Journal code, ONC, 8711562, ISSN: 0950-9232

1.26 ANSWER 22 OF 34 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC

- The tole of vascular endothelial growth factor (VEGF) in the pathophysiology of multiple myeloma (MM
- SO. Blood (November 16, 2000) Vol. 96, No. 11 Part I, pp. 836a. print Meeting Info 42nd Annual Meeting of the American Society of Hemato ogy Hematology San Francisco, Cal forma, USA December 01-05, 2000 American Society of ISSN 0006-4971

1.26 ANSWER 23 OF 34 BIOSIS COPYRIGHT 2002 BIOI OGICAL ABSTRACTS INC

- via the PI3-kinase and Akt protein kinase-B (PKB) signal pathway. VFGFR-1 (FIt-1) and VEGFR-2 (KDR) stimulate the proliferation of AVI. cells
- SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 301a, print. Hematology Meeting Info 42rd Annual Meeting of the American Society of Hematclogy San Francisco, California, USA December 01-05, 2000 American Society of Leaf-9000 NSSI
- 1.26 ANSWER 24 OF 34 BIOSIS COPYRIGHT 2002 BIOJ OGIC VI. ABSTRACTS INC
- 11 At GF binding domains on libronectin potentiate endothelial cell migration and differentiation by promoting the physical association of VFG1R-2(FLK-LKDR) with the integrin alpha5beta1
- SO. Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 36a. print Hematology San Francisco, California, USA December 01-05, 2000. American Society of Meeting Info 42 nd Annual Meeting of the American Society of Hematology ISSN 0006-4971
- 1.26 ANSWER 25 OF 34 BIOSIS COPYRIGHT 2002 BIOLOGIC VI. A SCIR ACTS INC. VFGF rescues hippocampal neurons from glutamate-induced toxicity through

Akt and ERK activation.

- SO Neuroscience Research Supplement, (2000) No. 24, pp. 851, print the 19th Annual Meeting of the Japanese Neural Network Society Yokohama. Meeting Info.: 23rd Annual Meeting of the Japan Neurosciet ce Society and ISSN: (921-8696. Tapan September 04-06, 2000
- TI VEGI -induced endothelial cell PAF synthesis, migration and proliferation: 1.26 ANSWI R 26 OF 34 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
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Louisiana USA November 12-15, 2000 ISSN 0009-7322. Meeting Info.: Abstracts from Scientific Sessions 2000 New Orleans

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The autophosphorylation of 1175-tyrosine residue on KDR FIk-

and MAP kinase pathway for DNA synthesis in vascular 1 (NEOF receptor-2) is essential for the activation of PLC-gamma

endothelial cells.

SO Teurnal of Submicroscopic Cytology and Pathology, (July, 2000) Vol. 32 No. 3, pp. 437, print.

Switzerland September 05-09, 2000 Meeting info.: NIth International Vascular Biology Meeting Geneva

ISSN: 1122-9497.

1.26 ANSWER 28 OF 34 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC

TI VEGE-induced PAF pathway synthesis by endothelial cells. Role of the P13 kinase

Cardiovascular Society Vancouver, British Columbia, Canada October 20-November 01/2000 Canadian F. pp. 143F, print. Meeting Info.: 53rd Annual Meeting of the Canadian Cardiovascular Society Canadian Journal of Cardiology, (September, 2000) Vol. 16, No. Supplement

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. ISSN: 0828-282N

Vascular endothelial growth factor (VEGF); axonal outgrowth and the

expression of the VEGF: Receptor FLK-1 in cultured peripheral gangha. SO: Society for Neuroscience. Abstracts. (1999). Vol. 25, No. 1-2, pp. 233. Meeting Info.: 29th Annual Meeting of the Society for Neuroscience, Part 1 Mami Beach Florida, USA October 23-28, 1999 The Society for Neuroscience ISSN: 0190-5295.

126 ANSWER 30 OF 34 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC

A new potential VE GF signaling pathway via phosphoin satide (PDF-kinase

SO Circu ation (Oct. 27, 1998) Vol. 98, No. 17 SUPPL., pp. 1327. Mee ing Info : 71st Scientiffe Sessions of the American Heart Association Texas, USA November 8-11, 1998 The American Heart Association

ISSN: 0009-7322

1.26 ASSAFR 31 OF 34 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC Vascular endothelial growth factor (VEGF) activates mitogen-

activated protein kinase (MAPK)

pathways through phosphatidylmositol 3^{\prime} (PI3) kinase in VEGFR-2

FIK-1 expressing hematopoletic cells

SO Blood, (Nov. 18, 1998) Vol. 92, No. 10 SUPPL, I PART 1-2, pp. 208A. Meeting Into 40th Annual Meeting of the American Society of Hematology Heamatology Minmi Beach, Florida, USA December 4-8, 1998 The American Society of

[264-9000 NSS]

1.26 ANSWER 32 OF 34 CAPITUS COPYRIGHT 2002 ACS

11 Signal transduction of vascular endothelial growth factor (NEGF) receptors, Elt-1 and SDR Elk-1

SO International Congress Series (1999), 1175(Tissue Engineering for Therapeutic Use 3), 25-33

CODEN FXMDA4. ISSN 0531-5131

126 ANSWER 33 OF 34 CAPILI'S COPYRIGHT 2002 ACS

11 8-(3-Oxo-4.5.6-triby droxy-3h-xanthen-9-yl)-1-naphthoic acid inhibits MAPK phosphorylation in endothelial cells induced by VEGF and bFGF

SO International Journal of Molecular Medicine (1998), 2(2), 211-215 CODEN LIMINEG, ISSN 1107-3756

1.26 ANSWER 34 OF 34 CAPITUS COPYRIGHT 2002 ACS

11 Tumor angiogenesis and Elt tyrosine kinase

SO Tanpakushitsu Kakusan Koso (1997), 42(10), 1470-1476 CODEN TAKKAI ISSN 003949450

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1.26 ANSWER 32 OF 34 CAPILUS COPYRIGHT 2002 ACS

ACCESSION NUMBER 1999 362303 CAPLUS

DOCUMENTALABLE 131 139567

Signal transduction of vascular endothelial growth

factor (VFOF) receptors, Flt-1 and KDR Flk-1

M. HIOR(S) Hiratsuka, Sachie, Ogawa, Sachiyo, Yabana, Naoyuki, Shibuya, Masabumi; Takahashi, Tomoko; Sawano, Asako;

CORPORATE SOURCE Maru, Yoshiro; Noda, Tetsuo; Yamaguchi, Sachiko Department of Genetics, Institute of Medical Science,

University of Tokyo Tokyo 108-8639, Japan International Congress Series (1999), 1175(Tissue

Engineering for Therapeutic Use 3), 25-33

CODI N. ENMID V4, ISSN 0531-5131

131 ISH R Elsevier Science B V

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VB. A review with 25 refs. including some of the authors' studies. VEGF Las been shown to be crucial for the physiol, and most of the pathol. receptors (VFOFR), Flt-1 and KDR Flk-1, the authors angrogeneses. To examine the signal transduction from the two VEGF

established NIHNIB cell lines overexpressing each of the VEOFRs. authors found that most of the mitotic signal was generated from KDR

kinase receptors, KDR FIk-1 utilizes the activation of FIk-1 However, unlike other representative tyrosine

> physiol angiogenesis, most likely regulating the levels of VEGF to an appropriate range in embryogenesis. are mediated by KDR type VI GII-like mol. (VEGE-E) which only binds and activates KDR ligand-binding domain of the FIt-1 is sufficient for the establishment of the flt-1 ty osine kinase-deficient mice suggest that the high-affinity attinity to VEGF but a much weaker tyrosine kinase activity. Studies on Flk-1. Flt 1 was found to earry a stronger binding signals towards endothelial cell proliferation and vascular permeability activation of Ras or PI3kinase pathway. The idea that most of the pos MAP kinase and DNA synthesis, but not or only weakly the phospholipase-C.gamma.-protein kinase C pathway for the stimulation of Flk-1 was confirmed by a novel

REFERENCE COUNT THERE ARE 25 CITED ROFFRENCOS AVAILABLE FOR

RECORD, ALL CITATIONS AVAILABLE IN THE REFORMAT

126 ANSWER 31 OF 34 BIOSIS COPYRIGHT 2002 BIOI OGICAL ABSTRACTS INC ACCESSION NUMBER 1999-98673 BIOSIS

DOCUMENT NUMBER PREV199900098673

Vascular endothelial growth factor (VFGF) activates

mitogen-activated protein

kinase (MA PK) pathways through

phosphatidylinositol 3' (PB) kinase in VEGFR-2 I'lk

1 expressing hematopoietic cells.

7. THOP(S) Wang, J.-F.; Groopman, J. E.,

CORPORATE SOURCE Div Experimental Med. Beth Israel Deaconess Med. Center.

Harvard Inst. Med., Boston, MA USA

SOURCE: pp. 208.1. Blood, (Nov. 15, 1998) Vol. 92, No. 10 SUPPL 1 PART 1-2,

Meeting Info.: 40th Annual Meeting of the American Society

The American Society of Heamatology of Hematology Miami Beach, Florida, USA December 4-8, 1998

ISSN 0006-4971

DOCUMENT TYPE Conference

LANGU AGIE English

1.26 ANSWER 21 OF 34 MEDIANE

ACCESSION NUMBER: 95124709 MEDIANE

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95124709 PubMed II): 7824260

A unique signal transduction from FLT tyrosine kinase, a

receptor for vascular endothelial growth factor VEGE

Seetharam I.; Gotoh N; Maru Y; Neufeld G; Yamaguchi S.

Shibuya M

MOIII M

CORPORATE SOURCE: Department of Genetics, University of Tokyo Japan

ONC. OGENE, (1995 Jan 5) 10 (1) 135-47

Journal code: ONC; 8711562 ISSN: 0950-9232

PUB COUNTRY: ENGLAND: United Kingdom

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199502

Entered STN: 19950223

Last Updated on STN: 20000303 Entered Medline: 19950214

VB | 11t-1 (fins-like tyrosine k hase-1), a receptor-type tyrosine kinase of Fli-1-NIHBT3 cells and endothelial cells. These results suggest that Flt-1 immediate early gene e-myc was not induced, whereas the e-fos was induced that FIt-1 encodes for a 180 kDa glycoprotein, binds VFGF with high sharing similar features with two other flt-family encoded proteins KDR mitogenic response in NIH3T3 fibroblasts. and the activation of the Fit-1 kinase is insufficient to trigger a kinase utilizes a unique signal transduction system in endothelial cells. signal transduction from many receptor kinases, was very weak in both kinases, tyrosine phosphorylation of She protein, an important adaptor for endothelial cells. Further, different from many other receptor tyrosine of PLC gamma and GAP complex on tyrosine in both type of cells. However, a of FIt-1 expressing NIH3T3 cells showed that VEGF induced phosphorylation the 1 lt-1 signal cascade in the environment of endothelial cells with that very weakly in Flt-1 expressing NIH3T3 cells. A comparative analysis of mitogenic response in transfected NIH3T3 fibroblasts. Interestingly, the affinity, undergoes autophosphorylation but does not generate any proliteration of vascular endothelial cells. In this study, we demonstrate receptor for Vascular Endothelial Growth Factor (VEGF) known to induce the FIK-1 and FIt-4, has been recently identified as a strong activation of VLVP kinases was observed only in

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SESSION SINCE FILE 1.01.11

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> L33 ANSWER 3 OF 13 11 Selective potentiation of paclitaxel (taxol)-induced cell death by 1.33 ANSWER 2 OF 13 SO ONCOGENE (2000 Mar 9) 19 (11) 1379-85. II Activation of the c-tos enhancer by the erk MAP kinase SO MOFFELL AR BILARM ACOLOGY (2001-Aug) 60 (2) 290-301. pathway through two sequence elements: the e-fos AP-1 and p62TCT sites kinase inh bition in human cancer cell lines. mitogen-activated protein kinase Journal ecde: ONC: 8711562 ISSN: 0950-9232 Journal code: NGR; 0035623, ISSN: 0026-895N http://www.naturesj.com/one/index.html/print.ISSN: 0950-9232. MEDILINE MEDILINE DUPLIC VIEL DUPLICATE 2

1.33 ANSW (R 4 OF 13 SO TOURNAL OF BIOLOGICAL CHEMISTRY (1997 Sep 12) 272 (17) 23438-9 TI Hypoxia induces e-fi activated protein kinase-dependent pathway Journal code: IIIV: 2985121R. ISSN: 0021-9258. os transcription via a **mitogen-**MEDILNE FELLY DITE TO

The Antioxidants as well L33 ANSWER 5 OF 13 SO FUROPEAN IOURNAL OF BIOCHEMISTRY (1997 Feb 15) 244 (1) 45-52. activation of extracellular-signal-regulated kinase 2 and Elk-1 lournal code/ FMZ; 0107600/ JSSN: 0014-2956. as oxidants activate e-fos via Ras-dependent VIEDLINE PHINDLIG 4

1.33 ANSWER 6 OF 13

T1 Selective response of ternary complex factor Sapla to different

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mitogen-activated protein kinase

SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF subgroups Journal code: PV3; 7505876, ISSN: 0027-8424 AMERICA, (1996 Oct 15) 93 (21) 11563-8

L33 ANSWER 7 OF 13 MEDILINE DUPLICATE 5

TI The p38 and ERK MAP kinase pathways cooperate to 1131 activate Ternary Complex Factors and e-fos transcription in response to UV

SO EMBO JOURNAL, (1996 Dec 2) 15 (23) 6552-63. Journal code: FMB: 8208664 [SSN: 026]-4189

TI The transcription factor TCF Elk-1. A nuclear sensor of changes in the L33 ANSWER 8 OF 13 CAPIT'S COPYRIGHT 2002 ACS

cellular redox status

). () 77-84 Acv. Exp. Med. Biol. (1996), 387(Biological Reactive Intermediates V).

CODEN: AEMBAP: ISSN: 0065-2598

T33 ANSWER 9 OF 13 III Induction of e-fos expression through JNK-mediated TCF Elk-1 phos ahorylation. DI PLICATE o

SO 1 MBO JOURN M. (1995 Dec 1) 14 (23) 5957-64 Journal code | 8208664, ISSN: 0261-4189

133 ANSWER 10 OF 3 MEDLINE

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11 Protein synthesis inhibitors reveal differential regulation of

stress-activated protein kinase pathways that converge on Elk-L mitogen-activated protein kinase and

SO MOLECULAR AND CELL ULAR BIOLOGY, (1995 Sep) 15 (9) 4930-8 Journal code: NOY: 8109087 ISSN 0270-7306.

L 33 ANSWER II OF IA

11 Ras MAP kinase-dependent and -independent signaling pathways target distinct ternary complex factors.

SO_GENES AND DEVELOPMENT (1994 Aug 1) 8 (15) 1803-16 Journal code FN3, 8711660 ISSN 0890-9369

LOS ANSWER 12 OF 13 AIRDRINE

DUPLICATE 8

[11] Involvement of growth factor receptors in the mammalian UVC response

SO CF11. (1994 Sep 23) 78 (6) 963-72 Journal code: CQ4: 0413066, ISSN: 0092-8674.

LSCANSWER BOF13 [11] The SRF accessors protein Flk-1 contains a growth factor-regulated transcriptional activation domain 711 DJ 1715

SO C111. (1993. Npt 23) 73 (2) 381-93.

Journal code: CQ4: 0413066 ISSN: 0092-8674

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The SRF accessory protein Elk-1 contains a growth

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tactor-regulated transcriptional activation domain

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Journal code, CQ4, 0413066 ISSN: 0092-8674

PUB COUNTRY United States

Journal, Article, (JOURNAL, ARTICLE)

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FILE SEGMENT Priority Journals

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AB. The Elk-1 and SRF transcription factors form a ternary

complex at the e-los serum response element (SRE). Growth factor mobility of the ternary complex, accompanied by increased stimulation rapidly induces a reversible change in the electrophoretic

phosphorylation of the Elk-1 C-terminal region and by the activation of a 42 kd cellular Elk-1

> phosphorylation of an SRE-binding protein efficiency of its formation. In vitro, MAP kinase activity in vivo is dependent on the integrity of the NINP region 'unations as a similar reduction in ternary complex mobility but has little effect on the partially purified p42 p44 NIAP kinase induces a activation by the SRF kinase sites. These findings directly link transcriptional multiple sites, which are also phosphorylated following growth phosphorylates the Elk-1 C-terminal region at factor stimulation in vivo. The Elk-1 C-terminal kinase Phosphorylation of Elk-1 in vitro by regulated transcriptional activation domain whose to the growth factor-regulated

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ANSWER LOF4

The effect of vascular endothelial growth factor on a rat model of traumatic arteriogenic erectile dysfunction.

SO JOURNAI OF UROLOGY, (2002 Feb) 167 (2 Pt 1) 761-7 Journal code | 0376374 ISSN | 0022-5347

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multiple mRNA sphee forms in SHR and WKY Vascular endothelial growth factor, tissue distribution and size of

SO CLINICAL AND ENPERIMENTAL PHARMACOLOGY AND PHYSIOLOGY SUPPLEMENT, (1995)

Journal code 10109; 7611484 ISSN: 0143-9294

ANSWER 3 OF A MEDI INT

11 Vascular endothelial growth factor: tissue distribution and size of multiple mRNA splice forms in SHR and WKY.

lddux 20 SO | CHNICAL AND ENPERIMENTAL PHARMACOLOGY AND PHYSIOLOGY, (1995 Dec.

| S]67-8

Journal code 1008, 0425076 ISSN 0305-1870

VSWIR FOLL

11 Biochemical characterization of two isoforms of FLT4, a VEGF receptor-related tyrosine kinase

SO ONCOGENE (1995 Mar 2) 10 (5) 973-84.

Journal code ONC, 8711562 ISSN 0950-9232.

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ANSWER LOF 12 MEDIANE

TI Controlled Expansion of Human Endothelial Cell Populations by

SO TRUMAN GENETI Cre-loxP-Based Reversible Immortalization. IERAPY. (2002 Jan) 13 (2) 321-34

Journal code: 9008980 [SSN:]043-0342

ANSWER 2 OF 12 VIEDLINE

TI Gene therapy-mediated expression by tumor cells of the angiogenesis inhibitor (1k-1 results in inhibition of neuroblastema

growth in vivo.

Journal code: IND-0052631, ISSN: 0022-3468 JOURNAL OF PEDIATRIC SURGERY, (2001 Jun) 36 (1) 30-6

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II The ang ogenesis inhibitor \$U\$416 has long-tasting effects on vascular end of eli il growth factor receptor phosphorylation and function.

SO CHNICAL CANCER RESEARCH, (2000 Dec) 6 (12) 4848-58 Journal ender C2II; 9502500, ISSN: 1078-0432.

1NSW :R 4 OF 12 MEDITAL

11 Characterization of (123)1-vascular endothelial growth factor-binding scintigraphy. sites expressed on human tumour cells; possible implication for tumour

SO INTERNATIONAL Journal code: GQU: 0042124. ISSN: 0020-7136. LIOURNAL OF CANCER, (2001 Mar 15) 91 (6 789-96

ANSWER 5 OF 12 MEDIANE

- TI Differential behavior of VEGF receptor expression and response to TNP-470 in two immortalized human endothelial cell lines.
- SO INTERNATIONAL Journal code: CNS ISSN: 1019-6439 JOURNAL OF ONCOLOGY, (2000 Sep) 17 (3) \$25-33

ANSWER 6 OF 12 MEDIENE

- TI | Lipecortin V may function as a signaling protein for vascular endothelial grewth factor receptor-2 Flk-1.
- SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1999 May 19)

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713-21

Journal code 9Y8, 0372516 ISSN, 0006-291N

ANSWER 7 OF 12 AIFINI INI

- 11 Multiple differentiation pathways of rat mammary stromal cells in vitro: dependent on hormonal and extracellular matrix stimulation acquisition of a fibroblast, adipocyte or endothelial phenotype is
- SO | DIFFERENTIATION, (1999 Jan) 64 (2) 91-101

Journal code 1:99, 0401650 ISSN 0301-4681.

ANSWER & OF 12 MI DI INE

- factor signaling The role of phosphatidylmositol 3-kinase in vascular endothelial growth
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Apr 9) 274 (15) 100 12-5 Journal code HIV, 2985121R JSSN 0021-9258

ANSWER 9 OF 12 VII-DI INE

- II Vascular endothelial growth factor induces VE-cadherin tyrosine phosphorylation in endothelial cells
- SO JOURNAL OF CLIT SCIENCE, (1998 Jul) 111 (Pt 13) 1853-65 Journal code HNK 0052457 ISSN: 0021-9533

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- 11 Inhibition of vascular endothelial growth factor (VEOF)-induced 7-encoded domain of VFGF165 endothelial cell proliteration by a peptide corresponding to the exon
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Dec 12) 272 (50) 3 582-8 Journal code HIV, 2985121R ISSN 0021-9258

ANSWER II OF 12 MEDI INE

- 11 Nuclear protein interactions with the human KDR flk-1 type-specific expression. promoter in vivo. Regulation of Sp1 binding is associated with cell
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Mar 28) 272 (13) 8410-6 Journal code HIV, 2985121R ISSN: 0021-9258

ANSWER 12 OF 12 VIEDLINE

- 11 FTK and LFRK-2 in developing kidney and nucrovascular endothelial assembly
- SO KIDNEY INTERNATIONAL SUPPLEMENT, (1996 Dec) 57 873-81 Journal code KVC: 7508622 ISSN 0098-6577.

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Center, Nashville, Tennessee, USA, CONTRACT NUMBER: DK34972 (NIDDK) L-K38517 (NIDDK)

1-K47078 (NIDDK) Journal code: KVC; 7508622, ISSN: 0098-6577. KIDNEY INTERNATIONAL SUPPLEMENT AT (1996 Dec) 57 S73-81

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VB. Eph 'amily receptor tyrosine kinases direct neuronal cell targeting bandling and intercellular aggregation activity, yet their role in progetitor cells of primitive microvasculature in a pattern similar to renal microvascular endothelial cells (HRMEC), and showed that EI K identified expression of ELK (Eph-like kinase) receptors in cultured human that of the VEGF receptor, flk-1. ELK LERK-2 and developing murine kidney. ELK and LERK-2 are expressed on endotnelial the ELK ligand, LERK-2, in HRMEC and in primitive vascular structures of response to the exogenous ligand, LFRK-2. Here we identify expression of mediates their in vitro assembly into capillary-like structures in mammalian kidney development has been unexplored to date. We recently

ureteric bud epithelium: FLK and LERK-2 expression persists in mature human umbilical vein endothelial cell (HUVEC) responses in an in renal-der ved endothelial cells may distinguish I ERK-2 from the angiogenic collecting ducts, glomeruli and arterioles. To explore whether of the renal microvasculature different vascular bed sources. ELK and its ligand, LERK-2, are spatially family receptors (ELK and Eck) are discriminated by endothelial cells from conditions that promoted HUVEC to assemble in response to capillary like structures in response to LERK-2, but not LERK-1, under vitro cap llary-like assembly assay. HRMEC endothelial cells assembled sources may distinguish among Eph receptor ligands, we compared HRMFC and Eck ligand, LERK-1 (B61), and whether endothelial cells from different **flk-1** antigens are also displayed on the branching and temporally coordinated in expression and may function in morphogenesis LERK-1 but not LERK-2. Therefore, responses mediated through specific Eph

L6 ANSWER 6 OF 12 MEDILINE

ACCESSION NUMBER 1999262163 VIEDLINE.

DOCUMENT NUMBER: 99262163 PubMed ID: 10329451

vascular endothelial growth factor receptor-2 FIK Lipocortin V may function as a signaling protein for

71.1.11C.K Wen Y; Edelman J L; Kang T; Sachs G

CORPORATE SOURCE California, 90073, USA Los Angeles VA Medical Center and UCLA Los Angeles, Membrane Biology Laboratory, Department of Medicine, West,

CONTRACT NUMBER DK41301 (NIDDK)

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SOLTECT BIOCHEARCAL AND BIOPHYSICAL RESEARCH COAMICATIONS.

Journal code, 9Y8, 0372516 [ISSN, 0006-291N May 19) 258 (3) 713-21

PUB COUNTRY Journal, Article: (JOURNAL, ARTICLE) United States

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FILE SEGMENT Priority Journals

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ナノコペン コイゴ Entered STN 19990712

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VIB. Binding of vascular endothelial growth factor (VEGF) to its receptor. VFGFR-2 (FIK-1 KDR), induces dimerization and

migration, and permeability enhancement. We explored the VEGF receptor library in yeast using the intracellular domain of rat VF GFR-2 as bait. for signaling proteins that relay the signals for cell proliferation, signaling pathway by performing a two-hybrid screen of a rat lung cDNA autophosphorylation of cytoplasmic tyrosine residues used as docking sites activation of the tyrosine kinase domain of the receptor, resulting in

Pretreatment of HUVEC with antisense oligodeoxyribonucleotide and hoocortin V. VEGF induced a rapid tyrosine phosphorylation of translated proteins confirmed the interaction between the VEGF receptor of VFGFR-2 was required for the interaction. Co-immunoprecipitation of hpocortin V in human umbilical vein endothelial cells (HUVEC). the yeast two-hybrid assay showed that the complete intracellular domain Two clones encoding lipocortin V were isolated. Subsequent studies with

mediated by VFGFR-2 be involved in regulation of vascular endothelial cell proliferation interacting with the intracellular domain of the receptor and appears to hpocortin V may function as a signaling protein for VEOFR-2 by directly and tyrosine phosphorylation of lipocortin V. Our results indicate that proliferation, which was accompanied by a decrease in protein synthesis (ODN) for hpocortin V significantly inhibited VEGF-induced cell

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The effects of RAS38 and antisense c-myc on cervical cancer cell lines with Figh expression of bel-2 gene.

Jul j 22 SO CHUNG-HUA CHUNG LIU TSA CHIII [CHINESE JOURNAL OF ONCOLOGA]. (200)

(4) 279-82. Journal code: 7910681. ISSN: 0253-3766.

ANSWER 2 OF 40 MEDITAL

II Activation of nuclear factor kappaB through the IKK complex by the human carcinoma cells. topoisomerase poisons SN38 and doxorubicin; a brake to apoptosis in HeLa

SO | CANCER RESEARCH (2001 Nov 1) 61 (21) 7785-91 Journal code: 2984705R. ISSN: 0008-5472

ANSWER 3 OF 40 MEDILINE

TI S phase dependence and involvement of NF-kappaB activating kinase to NF-kappaB activation by camptothecin.

SO BIOCLEMICAL PHARMACOLOGY, (2001 Sep 1) 62 (5) 603-16 Journal code: 010103 ISSN: 0006-2952

ANSWER 4 OF 40 MEDILVE

'II The human extomegalovirus US28 protein is located in endocytic vesicles and undergoes constitutive endocytosis and recycling.

SO MOLECULAR BIOLOGY OF THE CELL, (2001 Jun) 12 (6) 1737-49 Journal code: BAU: 9201390 ISSN: 1059-1524.

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Transcription factor AP-2 functions as a repressor that contributes to the

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II Cutting edge activation of HIV-1 transcription by the MIIC class II transactivator

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Journal code NTQ, 0326264 ISSN 0028-1298

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SO | 1 MBO JOURN M. (1999 Nov 15) 18 (22) 6370-84.

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11 Visualization of agonist-induced sequestration and down-regulation of a green fluorescent protein-tagged beta2-adrenergic receptor.

SO JOI RNALOF BIOLOGICAL CHEMISTRY, (1998 Jan 2) 273 (1) 322-8 Journal code; HIV: 2985121R, ISSN: 0021-9258

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SO_FIGCHEMICAL PHARMACOLOGY. (1997 Jun 1) 53+11) 1673-82 Journal code: 974; 0101032 JSSN: 0006-2952

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SO MOLECULAR AND CELLULAR BIOLOGY, (1997 Jul) 17 (7) 3833-40. Journal code: NOY: 8109087. ISSN: 0270-7306

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- SO JOURNAL OF VIROLOGY (1996 Jan) 70 (1) 641-6 Journal code | KCV | 0113724 | ISSN | 0022-538N

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- SO JOURNAL OF CELL SCIENCE, (1995 Apr.) 108 (Pt 4) 1617-27 Journal code HNK: 0052457 ISSN 0021-9533

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- SO MOJ FOUT AR AND CELLULAR ENDOCRINOLOGY (1991 Jun) 78 (1-2) 61-9 Journal code: F69; 7500844 ISSN: 0303-7207.
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- II Stable expression of human tissue-type plasminogen
- HeLa, WI-38 VA13 and KMS-5 activator regulated by beta-actin promoter in three human cell lines
- SO BIOCHIMICA ET BIOPHYSICA ACTA, (1991 Oct 8) 1090 (2) 216-22 Journal code: NOW; (0217513 ISSN: 0006-3002
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113 ANSWER LOFS MEDIANE

II. IIIV-1-trans-activating (Tat) protein: both a target and a tool in therapeatic approaches.

SO BIOCHEMICAL PHARMACOLOGY (1999 Nov. 15, 58 (10) 1521-8 Ref. 89 Journal code: 974; 0101032 [ISSN: 0006-2952]

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- KDR, is up-regulated by hypoxia.
- SO JOURNAL OF BIOLOGICAL CHEMISTRY (1997 Sep 19) 272 AS 23659-67 Journal code: HIV: 2985121R | ISSN: 0021-9258

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- [3] Nuclear protein interactions with the human KDR flk-1 premeter in vivo. Regulation of Sp1 binding is associated with cell
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 SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Mar 28) 272 (13) 8410-6
 fournal code: HIV: 2985121R-ISSN: ()()(21-9258)

LIS ANSWER FOR 5 AHEDLINE

- 11 Heterodimers of placenta growth factor vascular endothelial growth factor 1 K-1 7 DR Indothelial activity, tumor cell expression, and high affinity binding to
- SO JOURNAL OF BIOLOGIC VECHENISTRY, (1996 Feb 9) 271 (6) 3154-62 Journal code: HIV: 2985121R | ISSN: 0021-9258.

TIN ANSWER SOFS MINDLINE

- [11] Vascular endothelial growth factor and its receptors.
 SO: PROGRESS IN GROWTH FACTOR RESEARCH, (1994) \$ (1) 89-97. Fell of Journal code 1X6S: 8912757 ISSN 0955-2235

CONTRACT NUMBER CORPOR VIF SOURCE | Department of Surgery, Harvard Medical School, Bos on ACCESSION NUMBER: 96216393 NIEDLINE DOCUMENT NUMBER: 96216393 PubMed ID: 8621715 LIN ANSWER 4 OF 5 Journal code HIV, 2985121R, ISSN: 0021-9258 J.A. Wang Y: Fang F. Flanagan J G: Tsang M L Massachusetts 02115, USA and high affinity binding to FIK-1 KDR. growth factor. Endothelial activity, tumor cell expression, 3154-62 Heterodimers of placenta growth factor vascular endothelial JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 Feb 9) 271 (6) Cao Y., Chen H., Zhou L.; Chiang M.K., Anand-Apte B; Weatherbee MI DI INE PO1-CA-45548 (NCI)

FNIRY MONTH FILL SLGMENT INIRY DATE PUB COUNTRY Entered Medline 19960619 Journal, Article, (JOURNAIL ARTICLE) Last Updated on STN 20000303 nglish Entered STN 19960627 Priority Journals United States

AB. Here we show that the Escherichia coli expressed monomers of placenta growth factor (PLGF)129 and vascular endothelial growth factor (VEGF)165 can be re-folded in vitro to form PLGF VEGF heterodimers. The purified tyrosine phosphorylation of a 220-kDa protein, the expected size for the homodimers, but not PI GF129 homodimers, form complexes with membrane receptors. VI GF165 homodimers and PLGF VEGF heterodimers stimulate vem endothelial cells reveals that PLGF VEGF heterodimers and VEGF 65 bind to this receptor. Cross-linking of 1251-ligands to human umbilical these in vitro assays. We also demonstrate the presence of natural homodimers. In contrast, PLGF129 homodimers have little or no effect in heterodimers display 20-50-fold less mitogenic activity than VEGF165 initogenic and chemotactic effects on endothelial cells. However, PLOF YEGE recombinant PLGF VEGF heterodimers and VEGF homodimers have potent soluble Flk-1 KDR receptor, Pl GF129 homodimers fail to cell lines. While PLGF VFGF heterodimers bind with high affinity to a P1 GF VF GF heterodimers in the conditioned media of various human tu nor

> the format on of PLGF VEGF heterodimers in cells producing both factors hemodi ne's are unable to induce tyrosine phosphorylation of this protein. These data indicate that PLOF may modulate VEGF-induced angiogenesis by KDR receptor in human umbilical vein endothelial cells, whereas PLGF129

DOCUMENT NUMBER: THE VISIT RESORT ACCESSION NUMBER M. THOR , ournal code Ref: 61 Vascular endothelial growth factor and its receptors PROGRESS IN GROWTH FACTOR RESEARCH (1994) \$ (1) 89-97 Neufeld G: Tessler S: Gitay-Goren H: Cohen T. Levi B Z A68; 8012757 ISSN: 0055-2235 94257859 94257859 PubMed ID: 7515293 MEDLINE

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AB Vascular endothelial growth factor (VEGF) is a highly specific initogen for receptors, but the function of the VEGF receptors in these cells is receptors on the cell surface of vascular endothelial cells depends on the species, the best characterized is the 165 amino acid long form (VFGF165) unclear. Recently, the tyrosine-kinase receptors encoded by the flt and VEGF155 is a heparin binding growth factor, and its interaction with V1 GF growth of a variety of VEGF producing tumours. Of the various VEGE in tumour angiogenesis. Antibodies directed against VEGF can inhibit the produced by different including smooth muscle, luteal and adrenal cortex cells. VEGEs are also properties are transcribed from a single gene as a result of alternative species which differ in their molecular mass and in their biological permeabilization of blood vessels. Five types of VEGF mRNA encoding VEGF vascular permeability related to platelet derived growth factor (PDGF). It is also known as the caseu ar endothelial cells and an angiogenic factor that is structurally receptors. HeLa cells and human melanoma cells also express cell surface VEGE presence of heparin-like molecules. Several cell types which do not splicing. VEGEs are produced and secreted by several normal cell types KDR 11k-1 genes were found to function as VEGF165 proliferate in response to VEGF such as boyme corneal endothelial cells. tumorigenic cells, and appear to play a major role factor (VPF) because it efficiently potentiates the

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1 16 NNSWER 1 OF 28 MEDILLE

Antitumor activity of cytotoxic T lymphocytes engineered to target

SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED ケーノニノこ vascular endothelial growth factor receptors.

Journal code 7505876 ISSN 0027-8424 VMF RIC A (2002 May 14) 99 (10) 7008-14

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administration in adult rat brain Angiogenic and astroglial responses to vascular endothelial growth factor

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hemangiosarcoma cell line (ISO-HAS).

Journal code 8000462 ISSN 0340-3696 ARCHIVES OF DERM VIOLOGIC ALRESTARCH (2001 Jun) 293-6) 296-301

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by extracellular HIV-1 Tat protein Activation of endothelial cell initiogen activated protein kinase ERK(1.2)

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I To ANSWER GOF 28 AILULINE

Angrogenesis inhibitors in the treatment of lung cancer

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and vascular abnormalities.

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TI Fixtrace Juliar matrix protein 1 (FCMI) has angiogenic properties and is expressed by breast tumor cells.

SO FASEBJOURNAL Journal code: FAS; 8804484, ISSN: 0892-6638 (2001 Apr) 15 (6) 988-94

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extodifferentiation of the Dunning prostatic adenocarcinoma

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III Vascular endothelial growth factor induces nephrogenesis and vanculogenesis.

SO JOURNAL OF THE AMERICAN SOCIETY OF NECHROLOGY, (1999 October 10), 2125-

Journal code: A6H; 9013836, ISSN: 1046-6673

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11 Inhibition of hepatic stellate cell contraction during activation in vitro El Il tyrosine kinase receptor family. El T-L by vascular endothelial growth factor in association with upregulation of

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Journal code: 93 8: 0372516 ISSN: 0006-291N

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SO CANCER RESEARCH (1999 fan 1) 59 (1) 183-8 Journal code, CNF, 2984705R, ISSN: 0008-5472

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SO JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY, (1998 Nov.) 9 (115 1008-2004

Journal code Noll 9013836 ISSN 1046-6673

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SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCHLICES OF THE UNITED

S1.711.5 OF Journal code PV3, 7505876, ISSN 0027-8424 AMERICA, (1998 Jun 9) 95 (12) 7086-91

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Journal code: COR, 0077427 ISSN 0008-6363.

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Nascular endothelial growth factor receptor. SO | CFTT GROWTH AND DIFFFRF NTI VIION (1998 Ian) 9 (1) 49-58 Journal code AYII: 9100024 ISSN 1044-9523

LIG ANSWIR 22 OF 28 VITULIVE

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- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 May 16) 272 (20) 13390-6 11 Extracellular cleavage of the vascular endothelial growth factor 189-a nino acid form by urokinase is required for its mitogenic effect.
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- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Mar 14) 272 (a1) 7151-8 Journal code: HIV: 2985121R, ISSN: 0021-9258.

1.16 ANSWER 25 OF 28 MEDIANE

- TI LIeterodi mers of placenta growth factor vascular endothelial growth factor FIK-1 KDR Endothelial activity, tumor cell expression, and high affinity binding to
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 Feb 9) 271 (6) 3154-62 Journal code: HIV; 2985121R, ISSN: 0021-9258.

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- SO | CANCER RESEARCH, (1996 Mar 15) 56 (6) 1324-30. Journal code: CNF; 2984705R. ISSN: 0008-5472

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- SO INTERNATIONAL Journal code: [A7F] 9111627 [SSN: 0925-5710] JOURNAL OF HEMATOLOGY, (1995 Dec) 62 (4) 203-15

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- TI Placenta growth factor. Potentiation of vascular endothelial growth factor not to FIK-1 KDR. bioactivity, in vitro and in vivo, and high affinity binding to Flt-1 but
- SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Oct 14) 269 (41) 25646-54 Journal code: HIV; 2985121R, ISSN: 0021-9258.

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